ABC OF INTENSIVE CARE

MOHAMED SALEM
PROFESSOR OF MEDICINE
EMERGENCY MEDICINE UNIT
CAIRO UNIVERSITY

Contents

Monitoring of critically –ill patients.

- **Respiratory rate:**
  - Normal 10-15/min.
  - Tachypnea >20/min is an ealy sign of respiratory distress while slow breathing may be due to depression of respiratory center by drugs, increased intracranial tension or brain stem stroke.
- **Mean arterial blood pressure:**
  Equals
  diastolic B.P+ ⅓ pulse pressure.
  Normally = 90-100 mmHg A value < 55 causes cerebral and myocardial ischemia.
  Pulse pressure= systolic – diastolic B.P. It reflects stroke volume.
- **Capillary refill time:**
  Normal < 3sec. Prolonged in shock states.
- **Urine output:**
  Normal >0.5ml/hr/Kg. It measures renal blood flow.
- **Fluid balance:**
  - Weight chart
  - Charts of inputs (oral + I.V.) and outputs (urine + sweats).
  - Insensible water loss from skin is normally 500 -1000 ml/day increased by fever.
- **Core tempature:**
  - (rectal, tympanic probes) and skin temp.
- **Central venous pressure: C.V.P. :**

Monitored by a catheter inserted via internal jugular or subclavian vein with its distal end at upper right atrium.

Normal pressure 3-11cm water. Low CVP indicates hypovolemia, while high CVP does not exclude hypovolemia, as it may increase in right sided heart diseases, pulmonary embolism, emphysema, positive pressure mechanical ventilation.

The CVP is not sensitive or accurate measure of left ventricular function.

- **Invasive B.P. monitor :**
  Using arterial cannula measures intra-arterial B.P., used in shocked patient as cuff readings are falsely low.
- **ECG monitoring on screen:**
  - For heart rate and rhythm changes : monitors have alarm system when high or low rates are recorded .
  - Monitoring S.T. segment shift.
- **Arterial blood gases : ABG's:**
  - Arterial blood sample is taked by heparinized syringes and analysed immediately.
  - Normal ABG's

Arterial oxygen tension (PO_2)	: 90-100 mmHg.
Arterial Carbon dioxide tension (PCO_2)	: 40 mmHg.
PH	: 7.35-7.45
Plasma bicarbonate	22-26mmol/L
Arterial oxygen saturation (O_2 %)	: 95-100%.

  - Normal venous blood gases:

(PO_2)	40mmHg
(PCO_2)	45mmHg
(O_2 %)	70-75%

Pulse oximetry :

Bed side, safe, reliable measurement of percentage of arterial oxygenated hemoglobin (O2%).

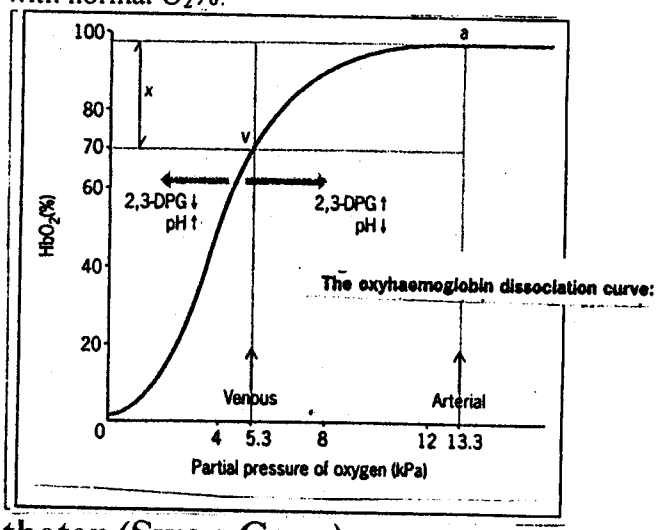
It is made up of a probe placed around the fingers emitting light at 2 wave lengths which when striking a pulsating artery can detect oxyhemoglobin % .

- Pulse oximetry is the fifth vital sign.
  - Continuous O_2 % monitoring can detect early hypoxemia in ICU patients.
    - Normal O_2% : 96 – 99%.
- It is used as a guide for O_2 therapy.
  - O_2 therapy at O_2 % 92% is unnecessary and exposes the patient to O_2 toxicity. It only markedly increase PO_2 but not O_2% (as O_2 dissociation curve becomes flat).
- O_2% is not a measure for oxygen content in arterial blood, which is a more accurate for aterial oxygenation.
  - Arterial O_2 content = O_2% X hemoglobin concentration.
- Pulse oximetry does not replace ABG's measurement. Dangerous hypercapnia, acidosis may pass undetected with normal O_2%.

Oxygen dissociation curve:

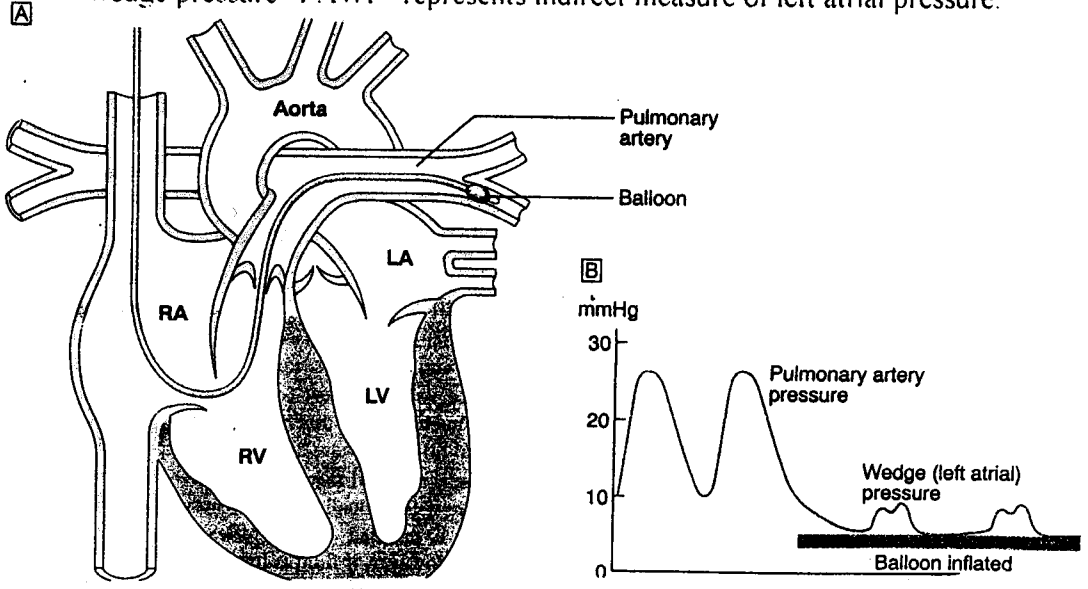
Shows relation between arterial PO_2 and O_2 %.

Modest fall in PO_2 is tolerated provided O_2 % is > 90%, however when PO_2 falls <60mmHg there is marked fall in O_2 % (respiratory failure).



Pulmonary artery flotation catheter (Swan Ganz):

Inserted through internal jugular vein and advanced to distal pulmonary artery branch, then the balloon at its tip is inflated and wedged preventing blood flow. Recording pulmonary artery wedge pressure "PAWP" represents indirect measure of left atrial pressure.



A pulmonary artery catheter. A There is a small balloon at the tip of the catheter and pressure can be measured through the central lumen. The catheter is inserted via a subclavian or femoral vein and advanced through the right heart until its tip lies in the pulmonary artery. When the balloon is deflated the pulmonary artery pressure can be recorded. B Advancing the catheter while inflating the balloon will 'wedge' the catheter in the pulmonary artery. In this position blood cannot flow past the balloon so the tip of the catheter will now record the pressure transmitted from the pulmonary veins and left atrium. This is known as the pulmonary artery wedge pressure or pulmonary artery occlusion pressure and provides an indirect measure of the left atrial pressure

Measures provided by P.A. catheter :

Parameter	Normal range
C.V.P	1-6 mmHg.
P.C.W.P.	6-12 mmHg
Cardiac output index	2.4 – 4L./min./m^2
Mixed venous oxygen saturation (from pulmonary artery)	70-75%
Oxygen delivery	1000ml/min.
Oxygen uptake (consumption)	250ml/min
Oxygen extraction ratio by tissues	20-30%

- O2 delivery represents cardiac output X arterial O2 content, the later is the product of O2 % saturation X haemoglobin in gms, so both low cardiac output and anemia decrease O2 delivery to tissues.
- O2 extraction ratio rises as O2 delivery diminishes or O2 consumption increases (hypermetabolic states, fever, anemia , seizures), to a maximum of 60% , above this tissue hypoxia and lactic acidosis occurs.

Central venous O2 Saturation

Measured Via C.V.P. catheter in right atrium is less invasive than P.A. catheter and slightly lower in value than mixed venous O2 sat%.

O2 extraction ratio can be calculated more easily: (arterial O2 % saturation – central venous O2 saturation)

Laboratory monitoring:

- **Biochemical screen**
  - Renal functions: urea, creatinine. Liver functions.
  - Electrolytes: sodium, potassium, chloride , bicarbonate, calcium, phosphate, magnesium.
  - Cardiac markers: CK-M.B., troponin.
- **Hematological screen:**
  - C.B.C
  - Coagulation screen
    - P.T. (12-16s). aPPT (30-40s.), thrombin time (12-16s), fibrin degradation products and D. dimer.
- **Septic screen:**
  - White cell count.
  - Blood culture
  - Blood lactate

Acute dyspnoea in emergency room

Causes:

Cardiac:

1- Acute cardiac pulmonary edema
2- Rapid arrhythmias (A.F).
3- Angina equivalent (due to transient L.V. failure).

Pulmonary:

1- Acute asthma
2- Pneumothorax
3- Pneumonia
4- Pulmonary embolism
5- Massive lung collapse.
6- Adult respiratory distress syndrome
7- Laryngeal edema

Others:

1- metabolic acidosis (uremia, ketosis)
2- psychogenic hyperventilation

Acute cardiac pulmonary edema

Causes:

(I) Acute L.V. failure due to:

1- rheumatic mitral valve disease with rapid A.F.
2- myocardial infarction.
3- Severe hypertension
4- Acute valve failure (mitral , aortic) due to:
   - Infective endocarditis
   - Aortic dissection
   - Papillary muscle infarction and rupture due to coronary occlusion
   - Prosthetic valve failure

(II) volume overload in acute renal failure.

Clinical picture: Fluid moves in interstitial lung tissue and alveoli.

- Patient is sitting up., agitated, sweaty, cyanosed and tachypneic.
- Coughing of pink, frothy, profuse sputum.
- Extensive lung crepitations and wheezes (cardiac asthma)due to bronchial mucosa congestion.

Investigations:

1- Plain X-ray chest
2- ECG: to detect rapid A.F. or underlying M.I.
3- A.B.G's: hypoxia with low or normal PCO_2.
4- Bed side ECHO to detect underlying cause.
5- Lab: Urea , creatinine CPK. MB, troponin

Alveolar pulmonary oedema. Typical 'bat's wing' pattern. The shadows are bilateral and maximal in the perihilar region

Treatment:

1- High flow 60-100% oxygen by mask. Persistent hypoxia is treated by mechanical ventilation.
2- Morphia 2.5-5mg I.V. acts as a venodilator
3- Nitroglycerine I.V. infusion 1-10mg /hr. is a venodilator reducing venous preload.
4- Furasemide 40-80mg I.V. /3-4hr. or by continuous infusion (20-80mg/hr).
5- Inotropics :
   Dobutamine infusion 5-20μg/Kg/min.
   May be combined with dopamine 2.5-5 μg/Kg/min.
6- Bronchospasm is relived by nebulized salbutamol and aminophylline infusion.
7- Intraaortic balloon pump
8- Dialysis in oliguric cases with hypervolemia.
9- Emergency valve repair in acute valve failure.

Acute Adult Respiratory Distress Syndrome ARDS .
"non – cardiac pulmonary edema"

ARDS is an inflammatory lung injury, alveoli are filled with inflammatory exudate (neutrophils and fibrin), so not responding to diuretics. Neutrophils release damaging proteolytic enzymes with capillary leak of exudate into alveoli. It heals by fibrosis.

Etiology:

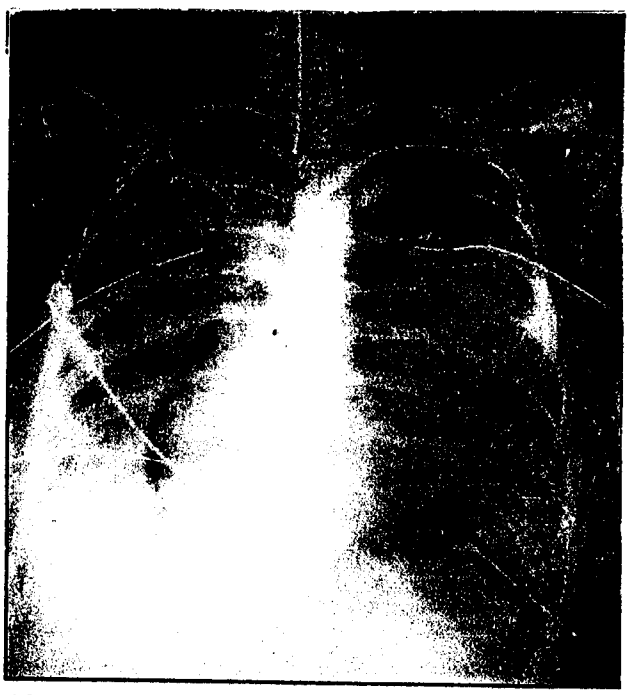
1- Sepsis
2- Pancreatitis
3- Increased I.C.P. (neurogenic pulmonary edema).
4- Multiple bony fractures
5- Direct lung injury by smoke inhalation, near drawning event or aspiration pneumonia

Diagnosis:

1- Progressive dyspnea and tachypnea
2- Refractory hypoxemia not responding to oxygen therapy.
3- CXR shows bilateral pulmonary infiltrate.
4- Absence of cardiac disease (clinical, by ECG and ECHO).

Treatment:

1- Low tidal volume (6ml/Kgm) mechanical ventilation.
2- Nitric Oxide inhalation
3- Steroids in late stages to prevent fibrosis



Chest radiograph of patient with severe adult respiratory distress syndrome. Note the widespread diffuse lung shadowing, the presence of four chest drains inserted due to recurrent pneumothoraces and the extensive subcutaneous emphysema and mediastinal air, resulting from barotrauma.

Acute severe bronchial asthma

Presented with a triad of wheeze, dyspnoea and cough.

Precipitants:

1- Infection
2- Exposure to known antigen.
3- Brittle asthma:

Type I: Wide PEF variations (morning dips) despite intensive therapy is a risk of severe attack

Type II: Sudden severe asthma in few minutes in a well controlled patient.

Assessment of acute asthma severity

Severity of an attack can be easily under estimated.

Acute severe asthma :

1- Tachypnea :R.R>25/min .
2- Tachycardia: H.R>100/min
3- Inability to speak.
4- PEF 30-50% of predicted to age, sex, height.

Life threatening asthma:

1- Silent chest (bronchi totally occluded).
2- Hypotension (systolic B.P. <100mmHg).
3- Arrythmia or bradycardia
4- Cyanosis. Pulse oximetry shows oxygen saturation below 92% despite O_2 therapy
5- Confusion, Coma.
6- PEF< 33% predicted

Near fatal asthma: Raised arterial PCO_2

Admission to ICU is needed.

Treatment:-

1- O_2 inhalation 60% or 15L/min. with high flow mask.
2- Nebulizer bronchodilators:
   a. Salbutamol 5mg every /15-30min . or continuous inhalation
   b. Ipratropium bromide 0.5mg/4-6hr.
3- Hydrocortisone 100mg I.V. /6hr.
4- Adequate hydration to prevent mucus plugging (2-3L/day).
5- Antibiotics if infection is present (yellow sputum, fever, neutrophilia, CXR: pneumonic shadow).

Monitoring progress clinically, and by pulse oximeter and P.E.F.

If no response or deteriorating:-

1- I.V. aminophylline

Loading dose 250mg slowly followed by infusion 0.5mg/kg/hr. with daily monitoring of serum levels to avoid toxicity (arrhythmias, seizures).

2- I.V. Salbutamol infusion 5mg in 500ml saline. Can cause tachycardia, tremors, hypokalemia.
3- Anasthetic help:

Inhaled anaesthetic agents (halothane, Ketamine, isoflurane) improve bronchospasm .

4- Mechanical ventilation

Acute Chest Pain In Emergency Department

Causes of acute severe chest pain:

1- Acute coronary syndromes:
The pain is heavy crushing retrosternal radiating into left shoulder, neck , jaw, arm and accompanied by sweating, vomiting and dyspnea.
2- Aortic dissection
3- pulmonary embolism.
4- Pneumothorax.
5- Pericarditis: the pain varies in intensity with movement and respiration.

Acute coronary syndromes

This includes:

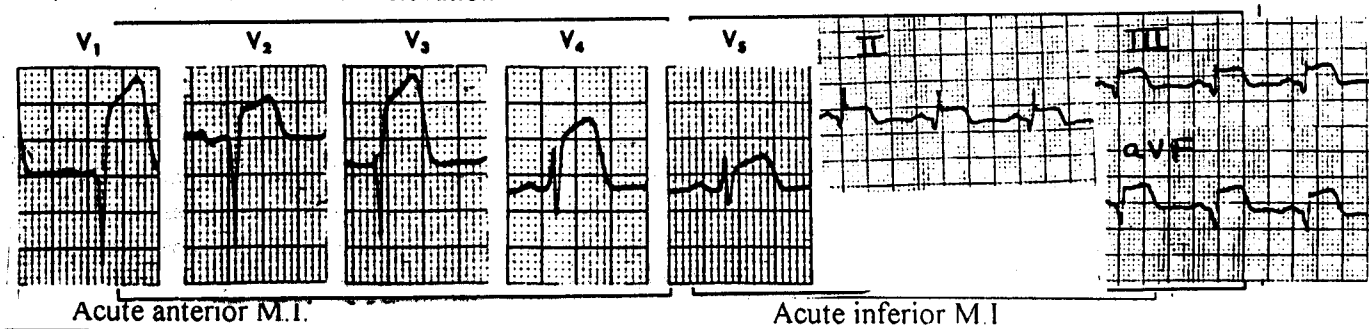
1) **Unstable angina (UA):**
   - Angina on rest, or sleep (nocturnal angina)
   - Rapidly worsening angina (crescendo)
   - New onset angina, with prolonged chest pain.
2) **Non- S.T segment elevation myocardial infarction (NSEMI)**:
   Previously caused subendocardial or non-Q myocardial infarction.
   Both are due to partial transient occlusion of coronary artery by platelet rich thrombus on top of ulcerated atheroma plaque. This is a dynamic process in which obstruction may increase or regress.
3) **S.T. segment elevation myocardial infarction (STEMI)**:
   (Q- myocardial infarction): due to complete occlusion of coronary artery leading to irreversible myocardial necrosis if not treated.

Early management of acute coronary syndromes

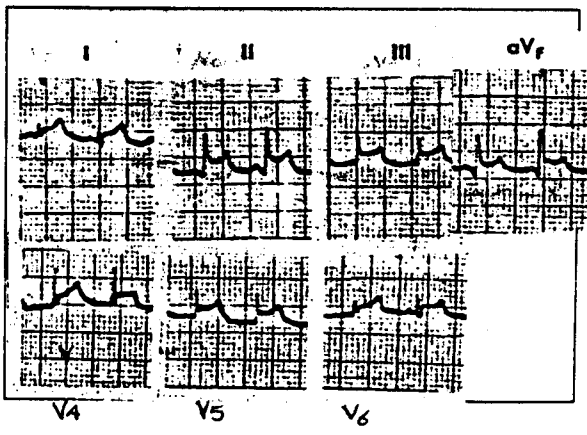
Initial triage:

Depends upon:

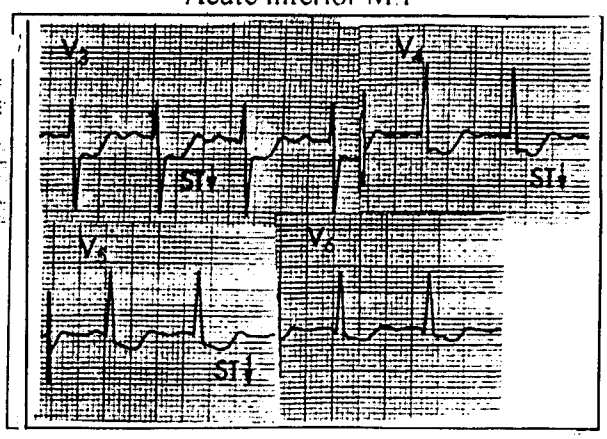
1- **ECG changes:**
In UA and NSEMI: ST depression. T wave inversion.
In STEMI: ST. elevation



Acute anterior M.I. Acute inferior M.I



Acute pericaditis. Diffuse concave ST seg. elevation



Non raised S.T. segement. M.I

2- Cardiac biochemical markers:

a- Creatine kinase (CK): rise at 6-4 hr for 48-72hr. it is non-specific as it is present in skeletal muscles and rise after I.M. injection.
CK-MB fraction is more cardio-specific

b- Cardiac troponins: is a sensitive marker of myocardial injury. Troponins rise at 4-6hr. for 2 weeks.
Serial daily estimations of CK.MB and troponins has more diagnostic value.

Early treatment of acute STEMI:-

1- Relieving chest pain:

1- Morphia 4mg I.V. slowly, repeated if needed with metoclopramide 10mg for vomiting. Morphia causes hypotension.
2- I.V. nitroglycerine 0.6-10mg/hr. infusion

2- Anti platelets:

Chewable aspirin 160-325mg inhibits platelets aggregation through inhibiting thromboxane production (anti-prostaglandin).
It reduces mortality, re-infarction and stroke

3- β- Blockers:

Decrease myocardial oxygen consumption (both negative inotropic, chronotropic effects) and arrhythmias. Atenolol or metoprolol (cardio selective) given initially I.V. *then* orally 50-100mg twice daily. Contraindication in C.O.L.D., bradycardia, hypotension and heart failure.

4- Angiotensin- converting enzyme inhibitors:

Inhibit cardiac remodeling (change in geometry of L.V.) contributing to post. infarction heart failure. It has survival benefit in patients with anterior M.I. and heart failure.
Captopril starting at low dose 12.5mg/8hr. and increase gradually.

5- Reperfusion therapy:

(a) Thrombolytic drug therapy:

Action: Activate plasma plasminogen to plasmin which breaks fibrin in thrombus.
Dose : Streptokinase 1.5million unit infusion over 1 hour.
Timing: Early (time loss is lives loss), within the first 12hr. of onset of chest pain. Benefit is greatest within the first 1 hr

Side effects of streptokinase:

- Fever, allergy (a bacterial protein)
- Formation of neutralizating antibodies decreasing its effect on subsequent use.

Recombinant tissue plasminogen activator (Alteplase):

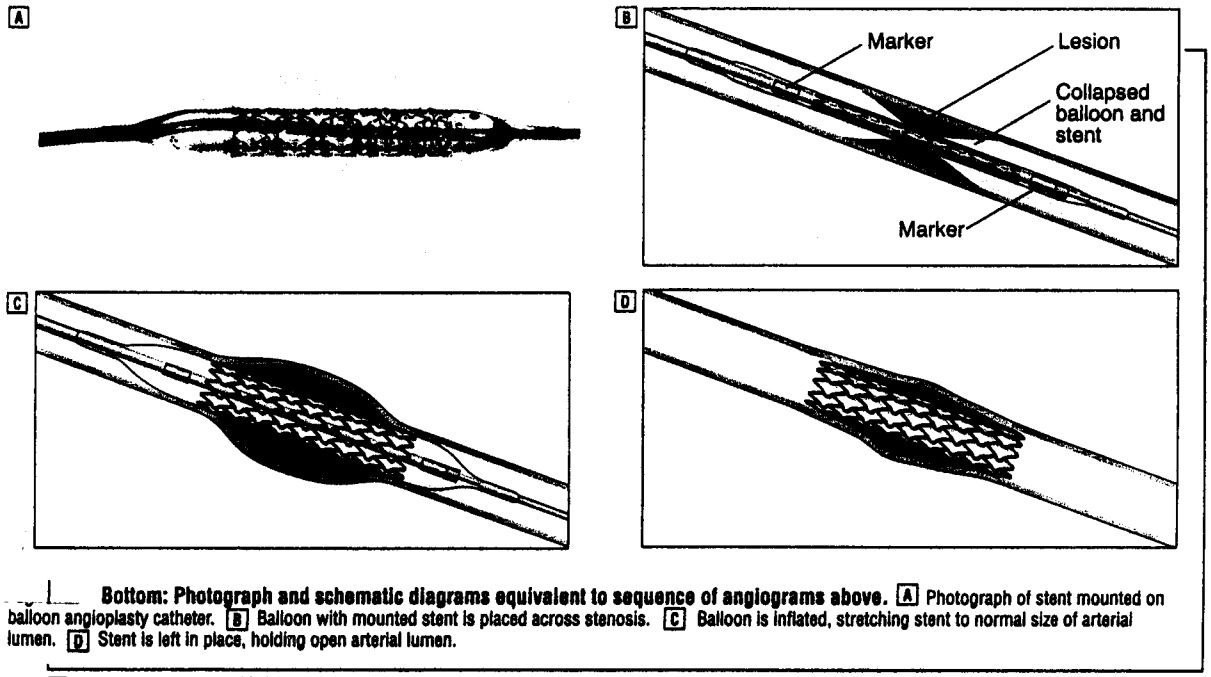
It does not produce allergy or antibodies and acts locally on fibrin in thrombus.
All thrombolytics can produce serious hemorrhage (ceberal hemorrhage in 0.5-1% of cases) and contraindicated in suspected aortic dissection, ischemic stroke, active peptic ulcer, major surgery, severe hypertension and pregnancy.

Heparin: Reduces the risk of:

- Reocclusion of coronary artery after thrombolysis
- Venous and arterial thromboembolism.
- Enoxaparin 1mg /Kg/12hr. S.C. for 5 days.

(b) Primary percutaneous balloon coronary angioplasty

Done within 90min has a lower mortality, reinfarction rate and stroke compared to thrombolytics



Bottom: Photograph and schematic diagrams equivalent to sequence of angiograms above. A Photograph of stent mounted on balloon angioplasty catheter. B Balloon with mounted stent is placed across stenosis. C Balloon is inflated, stretching stent to normal size of arterial lumen. D Stent is left in place, holding open arterial lumen.

Treatment of UA and NSTMI:

Urgent I.C.U admission as they may progress to Q- infarction or death occurs.

Beside I.V nitroglycerine and β- Blockers the following is used:

Oral antiplatelets:

Aspirin combined with clopidogrel 300mg initially then 75mg /day has additive effect. Clopidogrel (Plavix) inhibits ADP induced platelet aggregation.

I.V. antiplatelet

I.V. infusion glycoprotein IIb/ IIIa inhibitor tirofiban (Aggrastat) inhibit platelet binding of Glycoprotein IIb/ IIIa receptors to fibrinogen, which is the final common pathway of plalelet aggregation (superaspirin).

It reduces risk of complete infarction.

Heparin: Added to antiplatelets further reduces mortality.

Early coronary angiography with angioplasty or coronary by pass surgery is done in high risk patients or patients not stabilized medically.

Aortic dissection

Tear in intima of atherosclerotic plaque in aorta allows blood to burst into media splitting aorta into 2 layers and cause narrowing of true lumen. Hypertension is the most important cause

Clinical picture:

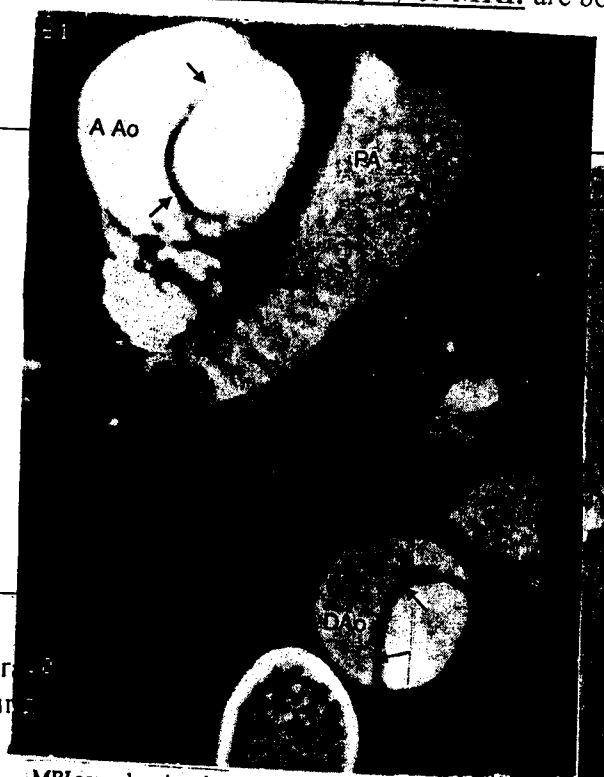
Type A: involves ascending aorta and extends to descending aorta.

- Tearing chest pain referred to back.
- Occlusion of branches of ascending aorta causes asymmetry of carotid and brachial pulses and B.P.
- Rupture into pericardium (tamponade) or left pleural sac
- Aortic valve dissection causing aortic regurgitation.

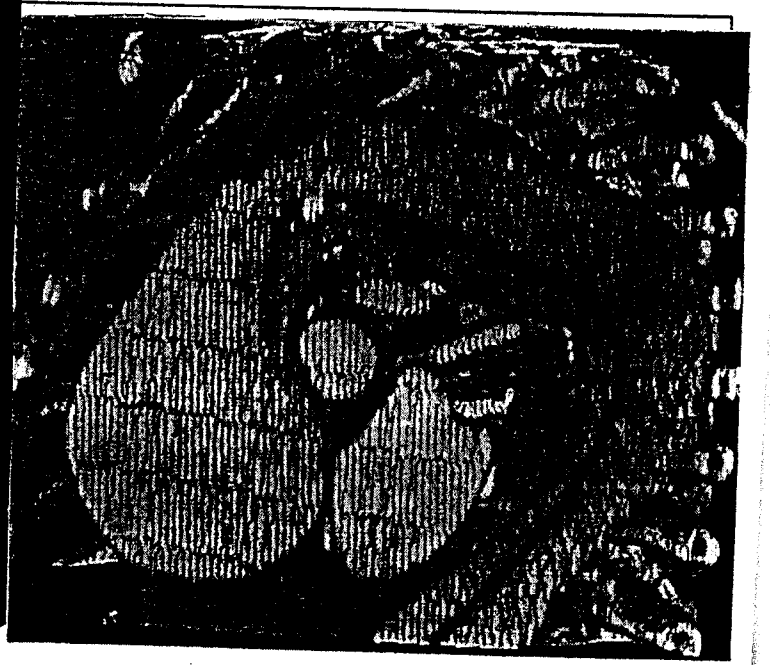
Type B: Spares ascending aorta, and causes occlusion of branches of descending aorta: mesenteric, renal, iliac arteries leading to acute abdomen, acute leg ischemia.

Investigations :

1- **CXR:** Shows broad upper mediastinum, left sided pleural effusion.
2- **ECG:** to exclude acute M.I.
3- **Transoesophageal ECHO:** Allows better visualization of aorta. It shows dilated aorta and intimal flap of dissection
4- **CT angiography or MRI:** are both highly specific replacing standard angiography



MRI scan showing the displaced intima in the ascending and descending aorta (arrows). AAo, ascending aorta; DAo, descending aorta; PA, pulmonary artery.



Thoracic aorta dissection. *Section through a 3D reconstruction of a type A thoracic aortic dissection. Both the true and false lumens can be seen extending from the aortic root to the descending aorta.*

Mangement:

Mortality is 1% per hour, in type A.

- Morphia for pain.
- Labetalol (combined alpha and beta blocker) to maintain systolic B.P. <120mmHg.
- Emergency surgery in type A with:
  - a- Replacing ascending aorta by Dacron graft.
  - b- Percutaneous endoluminal repair by stent graft placed from femoral artery.
- Type B: can be treated medically.

Shock

Definition: Impaired tissue perfusion and oxygen delivery.

Shock is not synomyous with hypotension, and should be treated before fall of B.P.

Causes :

1- **Hypovolaemic**: Blood or fluid loss
2- **Cardiogenic :** Severe heart failure
3- **Obstructive:**
   a. Major pulmonary embolism
   b. Cardiac tamponade
   c. Tension pneumothorax
4- **Anaphylactic.**
5- **Septic.**

General features of shock

1- Hypotension (systolic B.P<100mmHg, mean B.P. <60Hg)
2- Narrow pulse pressure below 30mmHg reflecting low stroke volume.
3- Tachycardia >100/min
4- Tachypnea
5- Oliguria (urine <30ml/hr)
6- Cold sweaty skin
7- Delay capillary refill sign > 3 seconds
8- Drowsiness, confusion, irritability.
9- Lactic acidosis due to tissue hypoxia
10- Multi-organ failure.

Initial assessment of shock should be urgent and rapid as recent data shows that early resuscitation improves survival

Hypovolemic shock

Causes:

1- **Haemorrhage:**
   a. External
   b. Concealed e.g retroperitoneal from leaking aortic aneurysm
2- **Fluid loss from:**
   a. G.I.T: vomiting, diarrhoea., ileus
   b. Renal : diuretics, diabetic ketosis
   c. Skin : burns
3- Extravascular fluid (third space) fluid sequestration: Pancreatitis,

Diagnosis:

Loss of up to 1 liter of extracellular fluid in adult is compensated by venoconstriction and may not cause any signs.

Loss of more than this causes:

- Postural hypotension (fall of systolic B.P by > 20mmHg). and tachycardia upon standing for 1min . Direct intraarterial .B.P. measument is more accurate in hypovolemia
- Very low C.V.P. with collapsed peripheral veins (difficult to cannulate).
- Loss of skin turgor (elasticity) due to interstitial fluid loss
- Raised plasma urea (pre renal).

- Hematocrit (packed red cell volume) and hemoglobin concentration are normal early in acute blood loss due to proportional loss of plasma and red cells and fall only after I.V. fluid therapy.

Treatment :

I.V. fluid therapy:

a) Given quickly in minutes to prevent acute tubular necrosis and tissue damage. Fluid are given through short wide bored peripheral cannulae as this ensures large volumes given quickly

b) Blood is not used for early resuscitation in acute blood loss, due to its high viscosity preventing rapid infusion

c) Over transfusion may result in pulmonary edema, which is more likely to occur in critically-ill patients because of their low plasma osmotic pressure (hypoalbuminemic) and associated adult respiratory distress syndrome. Severe hypovolemia also causes venoconstriction, over rapid correction does not give time to reverse

Crystalloid fluids should be replaced initially 2-3L. over 20-30min then at a rate of 1 liter /4-6hr. guided by clinical assessment, C.V.P and PCWP (not more than 15-18mmHg).

d) **Types of fluids used:**

- **Crystalloids:** Saline 0.9%, Ringers lactate
- **Colloids :** as polygelatin solutions, heta starch "starch polymer", dextrans and human albumin

  Colloids are three times more effective than crystalloids in increasing blood volume and cardiac output, as they remain in vascular space, while saline expands mainly interstitial space as it is rapidly lost from blood.
  Saline is therefore preferred in dehydration states. Albumin is not used as routine volume replacement fluid and only used in hypoalbuminemic cases.
- **Clucose 5%:** is ineffective plasma expander, and used only in pure water loss (diabetes insipidus). Its routine use in ICU patients lead to hyperglycemia which has adverse effects (increased risk of infection, aggravation of ischemic brain injury). Many of ICU patients are diabetics or have stress hyperglycemia.
- **Blood :** is used in hemorrhagic shock. Restrictive strategy of blood transfusion (hemoglobin 7-9gm/dL) is as effective and safer than liberal strategy (Hb 10-12g/dl).
  Blood transfusion has many risks (immune suppression, disease transmission). Over transfusion of large volume of stored blood rapidly can cause:-
  - Hypothermia (blood is stored at 4°C).
  - Coagulopathy and thrombocytopenia (stored blood has low plaletets and no cloltting factors).
  - Hyperkalemia
  - Hypocalcaemia (citrate in blood binds calcium).
  - Acute lung injury.

  Patients with hemorrhagic shock can tolerate very low Hb provided they are normovolaemic

Cardiogenic shock

Definition :-

Very low cardiac output and hypotension with elevated PCWP>18mmHg, Mortality>50%.

Causes:-

1- Extensive myocardial infarction is the main cause, especially if complicated by papillary muscle rupture (acute M.R.),right ventricular infarction.

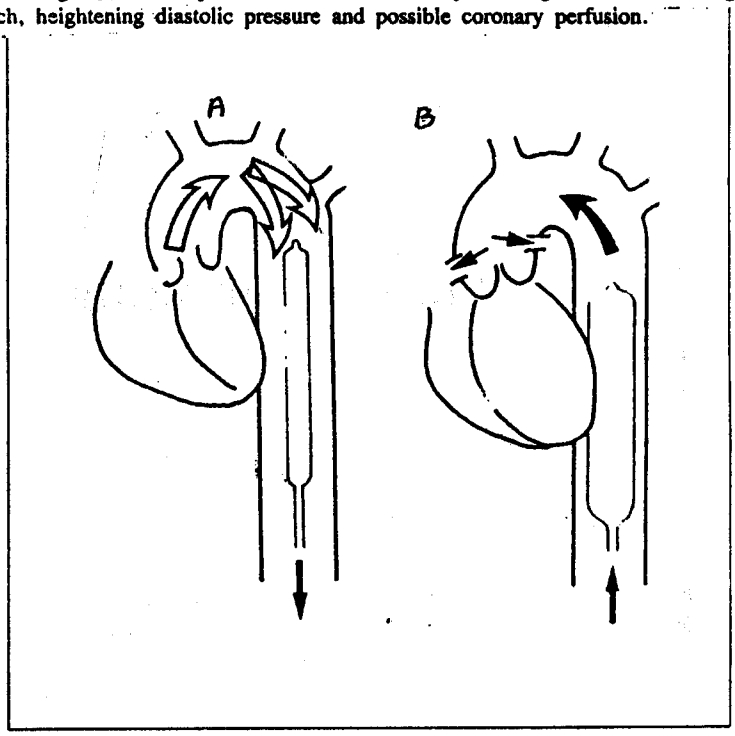
2- Other causes include severe cardiomyopathy and rheumatic valvular diseases

Diagnosis:

Depends upon:

- ECG: shows evidence of acute M.I.
- Cardiac biochmical markers raised in M.I.
- CXR: may show pulmonary edema.
- Bedside ECHO
- C.V.P: is raised
- Pulmonary artery catheter: shows reduced cardiac output, raised PCWP, and low mixed venous O_2 saturation reflecting increased tissue O_2 extraction

A Balloon deflation before systole, allowing antegrade volume flow from the aortic arch (systolic unloading). B, Counterpulsed diastole mechanically boosting volume flow retrograde to the aortic arch, heightening diastolic pressure and possible coronary perfusion.



Treatment: (in acute M.I)

1- Oxygen therapy if hypoxemia is present, by mask or mechanical ventilation

2- D.C shock for tachyarrythmias

3- I.V. insulin infusion if diabetic.

4- Correction of any hypovolemia (due to vomiting, excess sweating)by I.V. fluids.

5- **Positive inotropics:**

To raise blood pressure and coronary perfusion

a. **Dobutamine :** β_1. cardiac adrenergic stimulant 2-20μg/kg/min I.V infusion side effect:

- Tachycardia
- Arrythmias
- Tolerance

b. **I.V. Milrinone :** phosphodiesterase enzyme inhibitor with a vasodilator effect.

6- **Vasopressores :**

a- dopamine: in moderate dose (2-10μg/kg/min) it acts on β_1. cardiac receptors "positive inotrope". At doses > 10 μg/kg/min. it is vasoconstrictor (α receptor action).

b- Norepinephrine: is a potent vasoconstrictor used in refractory hypotension (2-4μg/min. IV. Infusion)

7- **Intra aotric balloon counter pulsation pump :**

A catheter with a balloon at its tip is inserted percutaneously through femoral artery under X-ray to lie below aortic arch. The balloon is infilated in diastole to increase aortic diatolic pressure, thus increaseing coronary and cerebral blood flow. It is only a temporary support.

8- Early revascularization (angioplasty or surgery) of infarction related artery decreased mortality if done within 18hr.

9- Corrective surgery for acute M.R or V.S.D. complicating infarction

Obstructive shock

Pulmonary Embolism (P.E.)

P.E. accounts for 5% of in-hospital mortality. The majority of P.E. arise from lower limb deep venous thrombosis (D.V.T), and a risk factor for D.V.T is present in almost all cases in the form of:

- Major surgery or post- partum
- Congestive heart failure
- C.O.L.D.
- Stroke
- Fractures
- Malignancy
- Pregnancy.
- Cases without identified risk factor for D.V.T. may have thrombophilia.

Clinical features:

Depends on size of embolus.

Acute massive embolus:

Symptoms:

- Central chest pain mimiking M.I.
- Severe dyspnoea
- Syncope

Signs:

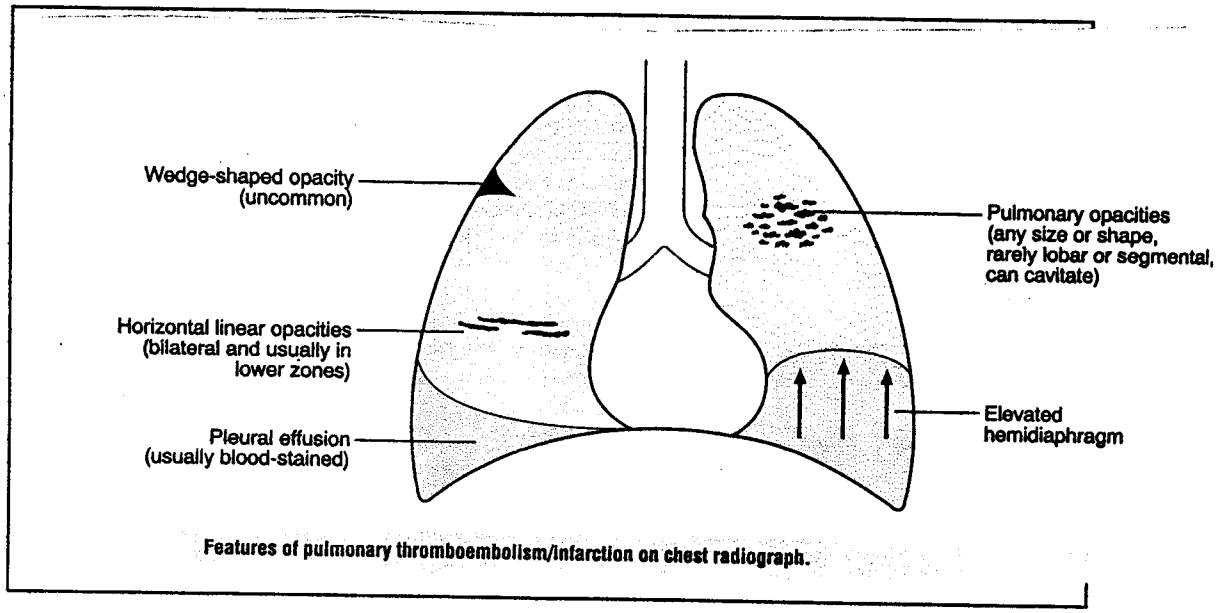
- Acute rightsided heart failure with congested neck veins, right ventricular gallop and tricuspid regurg.
- Tachycardia and hypotension.
- Cyanosis
- A.F. may be the presenting sign.

Acute Medium sized embolus:

Occluding segmental branch of pulmonary artery causes pulmonary infarction: fever, hemoptysis, pleuritic chest pain and blood stained pleural effusion, resembling pneumonia.

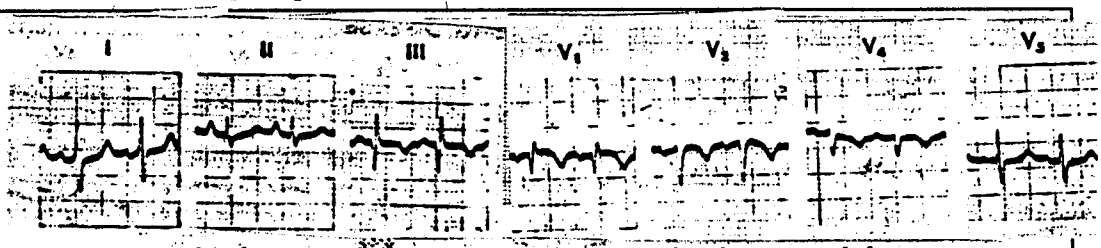
Investigations:

CXR: may be normal. Normal CXR in acutely dyspneic hypoxic patient should raise suspicion of P.E. It may show non-specific variety of findings:



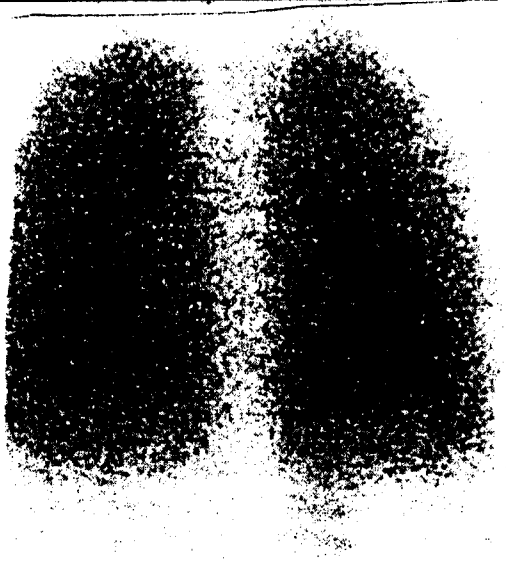
Features of pulmonary thromboembolism/infarction on chest radiograph.

- **ECG:** Shows non-specific findings as right ventricular enlargment or right bundle branch block or S_1 Q_3 T_3 pattern.

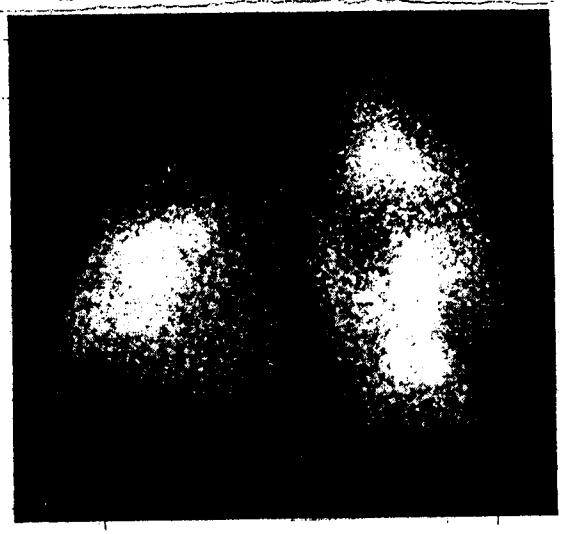


ECG shows several features occasionally seen with pulmonary embolism. Note sinus tachycardia, S wave in lead I with Q wave in lead III (S_1Q_3 pattern), as well as poor R wave progression and T wave inversions in chest leads resulting from acute right ventricular strain.

- **ABG's:** Shows reduced PO_2 , and normal or low PCO_2 (due to hyperventilation)
- **D. Dimer test:** it is a degradation product of cross- linked fibrin in the thrombo- emboli released in blood by endogenous fibrinolysis. Negative test is a useful screening test to exclude P.E., however positive test is non-specific.
- **Isotopic perfusion lung scan.**
  Shows perfusion filling defect. Normal scan excludes P.E. in patients with pre-existing chronic lung disease with abnormal CXR., this image is not useful.



Normal radionuclide perfusion scan using ^{99m}Tc-labelled macroaggregates of albumin. Posterior view.



Technetium 99m (TC 99m) labelled albumen macrosphere perfusion scan. A wedge-shaped perfusion defect is evident in the right lower lobe and there are extensive perfusion defects in the radiographically normal left upper and lower lobes.

- Duplex ultrasound of lower limb veins may detect D.V.T.

Echocardiography:

Shows acute dilatation of right ventricle and may be thrombus (embolism in transit).

Spiral computerized tomography C.T. angiography:

Can detect both P.E. and pelvic, lower limb thrombi.

Pulmonary angiography: is the gold standard test, being invasive it is less used now.

Spiral CT with contrast showing filling defects in pulmonary artery

Pulmonary angiogram showing filling defects in pulmonary artery

Management:

1- Oxygen therapy to raise arterial O_2 saturation above 90%.

2- Heparin therapy:

Thromboprophylaxis:

Low molecular weight heparin (LMW) enoxaparin 40mg S.C daily. In high risk ICU patients.

Therapeutic:

Enoxaparin 1mg /kg/12hr. SC for 5 days.

Heparin reduces mortality in P.E. by reducing propagation of clot and risk of further emboli.

LMW heparin is as effective as unfractionated heparin, and has less incidence of both bleeding and heparin- induced thrombocytopenia and no need for laboratory anticoagulant monitoring.

3. Thrombolytic therapy:
   Used for cases with shock and acute right ventricular failure.
   Alteplase 100mg infused over 2hr.
4. Warfarin is used for:
   - 3-6 months for cases with reversible risk factors and for life in recurrent D.V.T. or thrombophilia .
   - Prothrombin time (INR) is kept 2 to 3. Recently low intensity warfarin therapy (INR 1.5-2) is used to decrease risk of bleeding

6. Inferior vena cava filter insertion (meshlike) to trap thrombi preventing them to reach pulmonary artery is used in cases with a contraindication to anticoagulants. They are inserted through femoral or jugular vein at bedside.

Pericardial tamponade

It is due to rapid or massive accumulation of fluid in pericardial sac compressing the heart. It can occur in:-

1- Any form of massive effusion e.g. malignant, tuberculous.
2- Hemopericardium: as in rupture aortic dissection or myocardium after infarction or the use of anticoagulants in uremic pericarditis

Clinical diagnosis:-

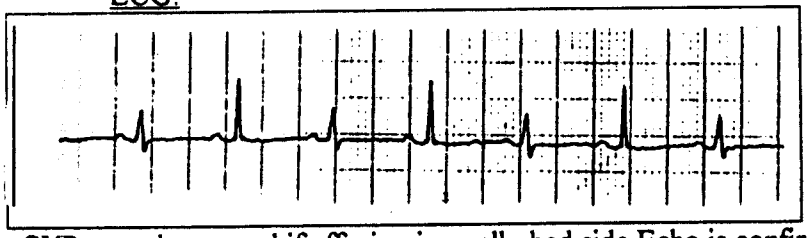
Symptom: Dyspnoea

Signs:

- Hypotension, tachycardia.
- Gross congestion of neck veins with paradoxical rise on inspiration "Kussmauls sign".
- Soft heart sounds.
- Pulsus paradoxicus: a large fall in systolic B.P. during inspiration "pulse may be impalpable".
- Friction rub may be present.

Investigations:

ECG:



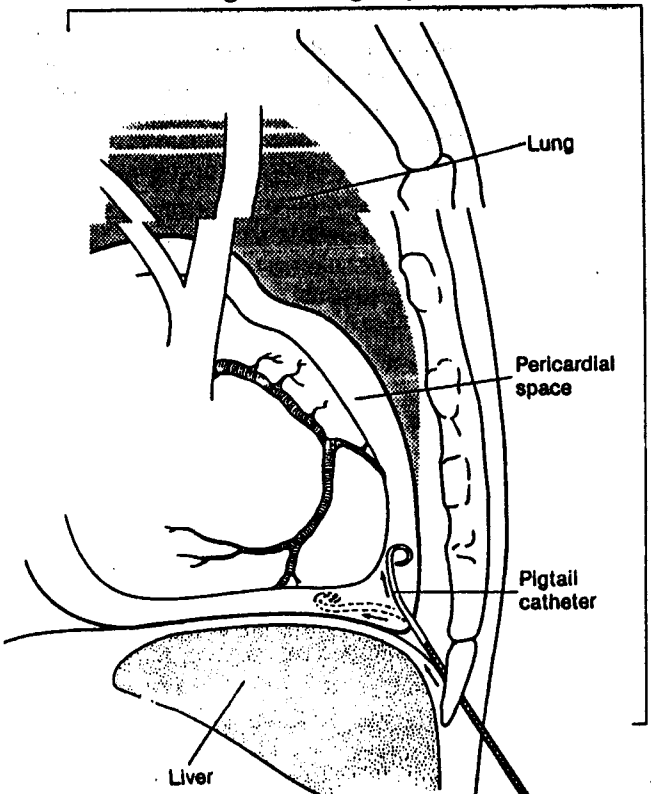
- Low voltage ECG.
- Alternating QRS in amplitude

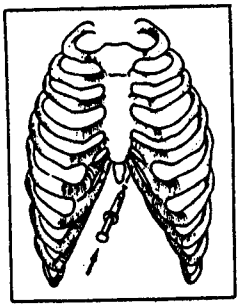
CXR: may be normal if effusion is small. bed side Echo is confirmatory.

Treatment:- pericardial aspiration by inserting a needle below xiphoid process directed toward left shoulder guided by Echo.

A plastic cannula inserted over the needle helps in therapeutic drainage.

Surgical emergency treatment is needed in loculated effusion or hemopericadium.





Tension pneumothorax

Air in pleural sac with a valve mechanism allowing air to be sucked in inspiration but trapped in expiration.

It occurs from rupture of emphysematous bullae or cyst especially in patients on ventilators or in asthmatics.

Clinical diagnosis:-

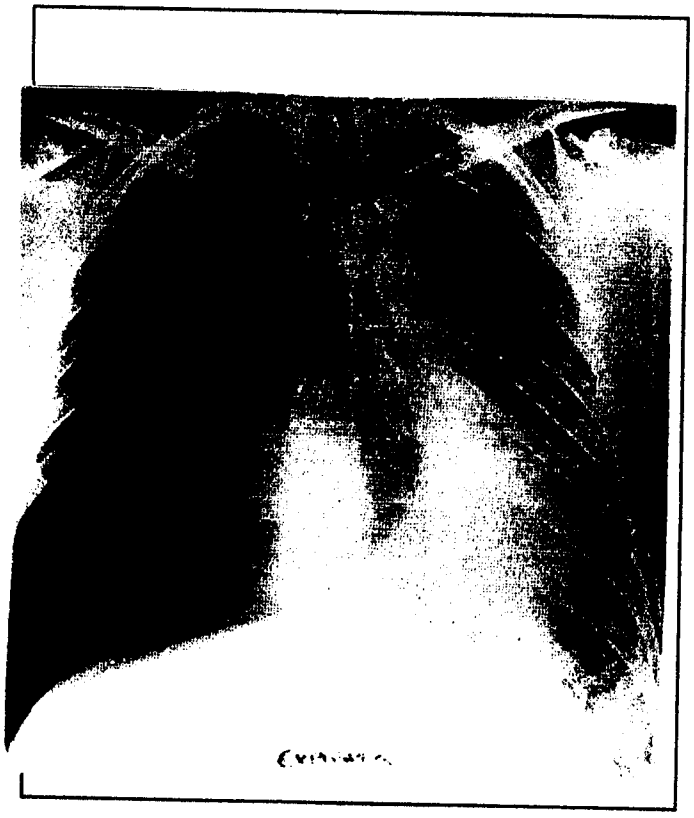
Symptoms:

- Sudden onset of pleuritic pain
- Acute dyspnoea.

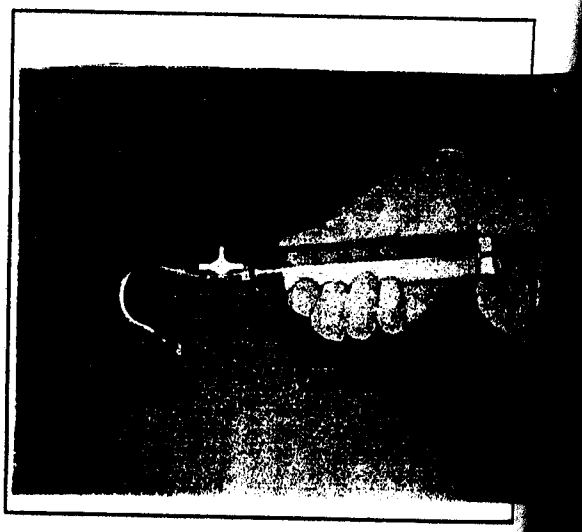
Signs

- Rise of intrapleural pressure impedes venous return causing hypotension, tachycardia .
- Lung collapse causes hypoxia and cyanosis.
- Resonant percussion and absent breath sounds.
- Urgent CXR.

Emergency treatment by pushing 3-4cm cannula 16 in second intercostal space (midclavicular or axillary) connected to a 3 way tap and 50ml syringe with air aspiration until lung reexpand. Followed by intercostal tube drainage under water seal.



Right tension pneumothorax.
Mediastinal shift with lung collapse.



Aspiration of tension pneumothorax in midaxillary line.

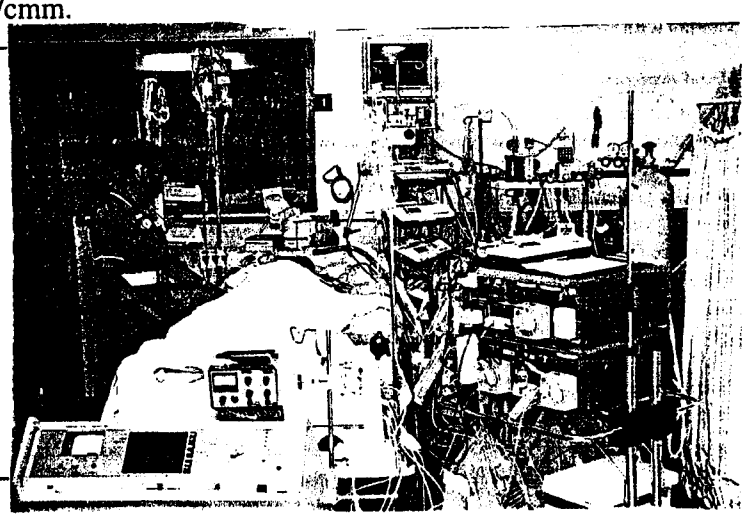
Septic shock

Definition of sepsis:-

Bactoraemia means presence of viable bacteria in blood.

Sepsis is documented infection causing systemic inflammatory response manifested by:-

- Fever * Tachycardia * Tachypnea.
- Polymorph leucocytosis > 12.000/cmm.



. A patient with multi-organ failure supported by haemodynamic monitoring, cardiac pacing, a counterpulsation aortic balloon pump, haemofiltration and nitric oxide therapy.

Septic Sock: accompanied by hypotension often unresponsive to fluid and vasopressors.

Multiple organ failure (MOF):

Means failure of more than one organ.

Etiology: the primary site of infection may be:-

1. Central I.V lines unchanged for> 4 days.
2. Nosocomial (hospital – aquired) pneumonia in intubated patients present in I.C.U. > days. Nasopharynx becomes colonized with gram –Ve bacteria causing pneumonia.
3. Intrabdominal abscess after abdominal operations or complicated intraabdominal sepsis or pancreatitis.
4. Other sites of infection:
   - Meningitis
   - Infective endocarditis
   - Severe kidney infections
   - Joints and bone infection
   - Post partum sepsis

Diagnosis of sepsis:

A. Fever

B. M.O.F.: caused by endo- and exotoxine of microorganisms in blood triggering inflammatory cascade. Many mediators (cytokines , adhesion molecules, endothelium released nitric oxide, plalelet activating factor, tissue factors triggering intravascular clotting) mediate sepsis syndrome.

M.O.F. is manifested by

1- Lungs: A.R.D.S.

2- Kidney: A.T.N.

3- G.I.T.

- Stress ulcer: hematemesis.
- Paralytic ileus.
- Acute hepatic injury

4- Nervous system: septic encephalopathy with altered level of consciousness.

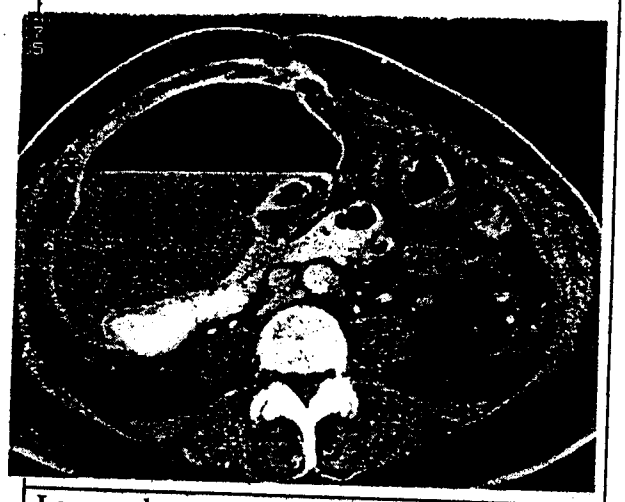
5- Blood : * D.I.C. * Thrombocytopenia

Investigations:

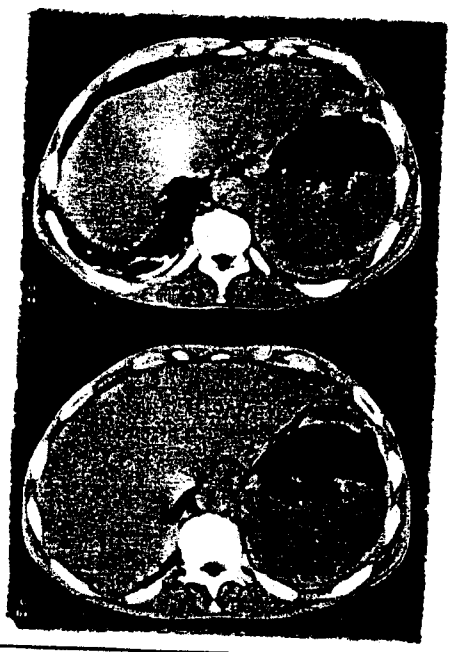
1- **Blood culture:** At least 2 blood samples 10ml each obtained from different veins. Positive in only 40-50%
2- Culture from primary site of infection
3- **C.V.P.**
   a. Is low due to increased capillary permeability with fluid loss in interstitial space (relative hypovolemia).
   b. High central venous O_2 saturation due to impaired O_2 extraction, as a result of toxic mitochondrial injury (tissue dysoxia).
4- **P.A. Catheter:**

Shows
- Low peripheral vascular resistance due vasodilatation (warm shock).
- High cardiac output (hyperdynamic shock).

5. **Radiological investigations:** for primary site of infection on urgent basis.



Large abscess with air fluid level and enhancing rim on contrast CT abdomen



Sub hepatic abscess with air- fluid level and contrast enhancing rim on CT abdomen.

Treatment:

1- Removal of primary source of infection, e.g. abscess drainage, I.V. catheter removal.
2- Imipenem (500mg/6h I.V.) with vancomycin (1mg/12hr) if methicillin resistant staphylococci is suspected .
3- Early resuscitation within the first 6 hr reduces mortality:
   a. I.V. fluid to correct hypovolemia
   b. Vasopressors: dopamine, nor-epinephrine and in refractory hypotension vasopressin or Glypressin I.V.
   c. Red cell transfusion if Hb <7 gm /dl. F.F.P. and plalelets transfusion in DIC.
   d. Mechanical ventilation in refractory hypoxia.
   e. Oral nutrition, prophylaxis against stress ulcer and D.V.T.
4- I.V. infusion of recombinant activated protein C reduces mortality when given early through preventing microthrombi and anti-inflammatory effect.
5- Hydrocortisone 50mg/6hr. I.V. for 7 days in resistant cases to vasopressors or in relative adrenal insufficiency.

Anaphylactic shock

Common causes: of systemic allergic reaction.

- Insect bites
- Drugs (e.g. antibiotics)
- I.V. anesthetics
- Foods
- Idiopathic 30%

Clinical features:

- Flushing, sweating, conjunctival injection.
- Urticaria
- Wheezy chest
- Hypotension.
- Angiodema of lips and tongue.
- Laryngeal obstruction (stridor)
- Diarthoea.
- Loss of consciousness.
- Arrythmias

Urgent management:

- Removal of further contact with allergen (bee sting)
- Upper airway patency with O_2 therapy
- I.V. saline to raise B.P.
- Adrenaline 0.3-1ml (1:1000) I.M./5-10min. reverses action of histamine within minutes
- Chlorphenamine antihistaminic I.M. or slow I.V.
- Hydrocortisone 100-300mg prevent late phase reaction
- Nebulized salbutamol

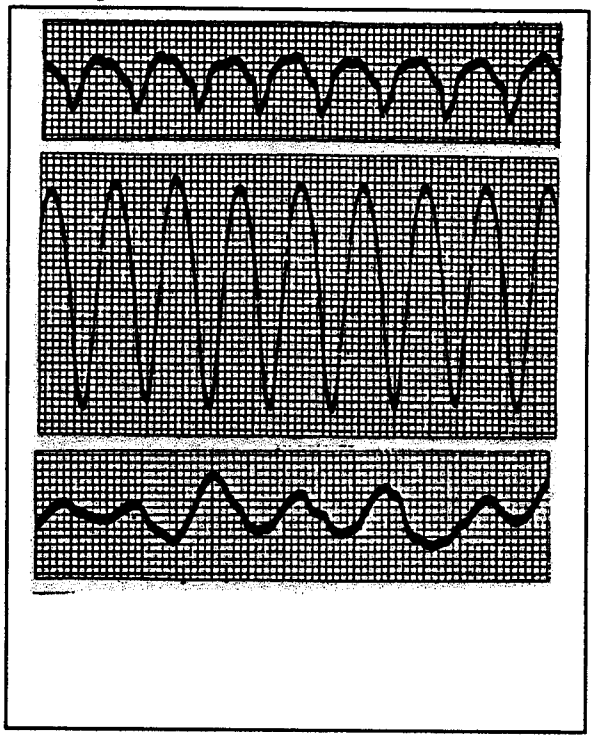
Cardiac Arrest

Types:-

1- Sustained ventricular tachycardia with hypotension and no pulse.
- Ventricular fibrillation (V.F) majority of cases 70-80%

2- Asystole

3- Pulseless electrical activity (PEA) (electromechanical dissociation)
No pulse, but ECG is present



The dying heart. V.T., ventricular flutter and fibrillation

Causes:-

1- Underlying structural heart disease, coronary heart disease accounts of majority (80%).

2- Functional causes include:-
- Shock states
- Electrolyte disturbances, acidosis
- Drug toxicity
- Heart failure
- Hypoxia

Cardiac arrest is abrupt, with complete loss of consciousness.

Prognosis: successful resuscitation of cardiac arrest depends upon:

1- Time from onset to C.P.R.:
By 5 minutes, survival rates are 25-30%

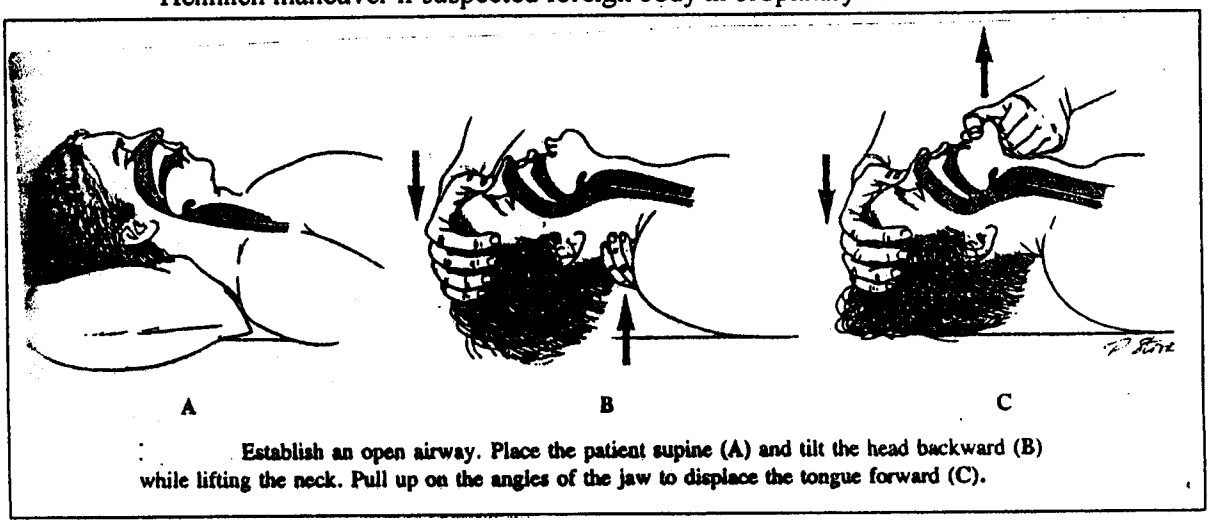
2- Type of arrest: V.T. and V.F. has best prognosis especially if occurring early after acute M.I. Asystole and PEA has bad prognosis.

3- Type of disease causing arrest:
Prognosis is good if correctable cause is present (e.g. hyperkalemia) or after acute M.I., while survival is poor < 10% in patients with terminal medical diseases or chronic advanced cardiac disease.
Death after successful resuscitation is due to anoxic encephalopathy, arrythmias or ventilator associated pneumonia.

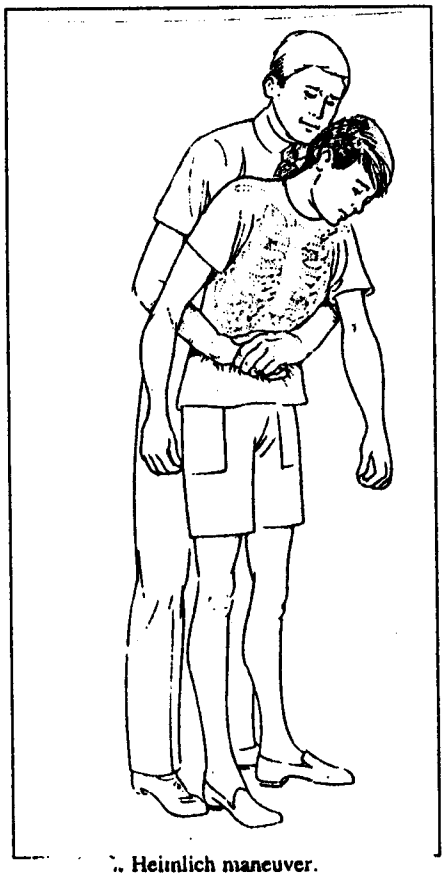
Treatment:

[Basic life support: cardiopulmonary resuscitation (CPR)]:

1- Clearing upper airways:
Head is tilted back and chin lifted with removal of foreign body from oropharynx.
Heimlich maneuver if suspected foreign body in orophharynx.



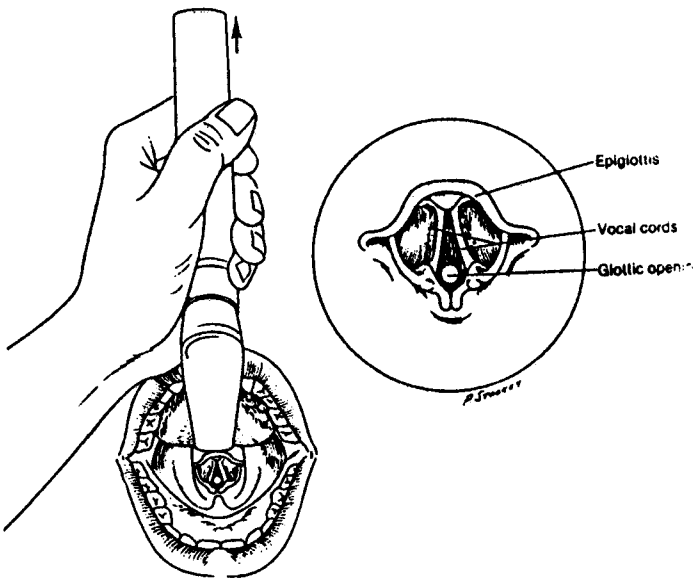
Establish an open airway. Place the patient supine (A) and tilt the head backward (B) while lifting the neck. Pull up on the angles of the jaw to displace the tongue forward (C).



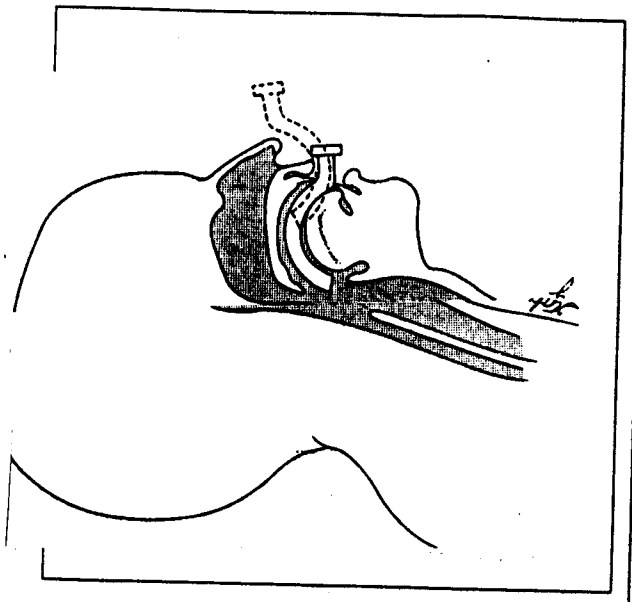
Heimlich maneuver.

2- Chest compression with out stretched arms over lower sternum (100 times/min.) to depress sternum 4-5cm with abrupt relaxation, allows the heart to maintain pumping

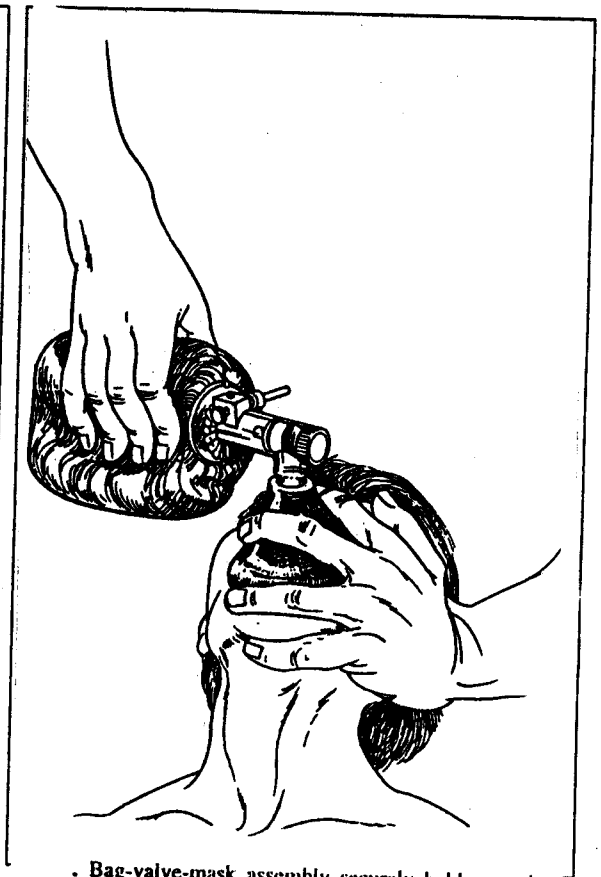
3- Mouth to mouth breathing or better by masked ambu bag to infilate the lungs twice in succession every 15 chest compressions.



Proper insertion of the laryngoscope to expose the glottic opening and landmarks.



. Proper insertion and position of the Guedel oral airway.



. Bag-valve-mask assembly securely held over the patient's mouth and nose with one hand, with compression of the bag with the other hand.

Advanced life support:

In VT and VF:

a. immediate electric defibrillation with 200J. to be repeated with 360J. up to 3 times if no response occurs .

b. if fails:-

- I.V. 1 mg adrenaline /3-5min. for 3 doses. Or vasopressin 40U. I.V once.
- Intubation and mechanical ventilation to reverse hypoxia.
- I.V. sodium bicarbonate 1mg/kg to reverse acidosis.
- Antiarrythmic drugs:
  - Amiodarone 150mg I.V. slowly followed by 1mg/min infusion.
  - Or lidocaine 1 mg /kgm I.V. bolus.
  - Or magnesium sulphate 1-2gm I.V in torsades de points V.T.

N:B: Calcium gluconte is not routinely used, only in acute hyperkalemia or hypocalcemia

Asystole /PEA:

- CPR
- Intubation with oxygen.
- Adrenaline 1mg I.V., atropine 1mg I.V. and sod. bicarbonate 1mg/Kg I.V. to be repeated
- Treatment of underlying causes such as :
  - Pericardial tamponade
  - Pulmonary embolism
  - Hypothermia
  - Hypovolemia
  - Tension pneumothorax
  - Hypoxia
  - Severe acidoisis
  - Hyper or hypokalemia.
- External cardiac pacemaker

Hypertensive emergencies

Severe elevation of B.P. (>220/120mmHg) Rapid rise of B.P. is more important in inducing organ damage

Classification.

1- Hypertensive emergency : with acute target organ damage.
2- Hypertensive urgency : No target organ damage.

Target organ damage.

1- **Brain**

Hypertensive encephalopathy: caused by cerebral edema, presenting with headache, vomiting, seizures, coma and focal neurological signs. Fundus shows papilloedema. An urgent CT brain is mandatory to exclude cerebral hemorrhage, subarachnoid hemorrhage.

2- **Cardiovascular:**

- Chest pain M.I. (diagnosed by ECG, cardiac markers)
- Acute heart failure, pulmonary edema (CXR).
- Aortic dissection.

3- **Renal:** Acute renal failure

4- **Fundus:** Retinal hemorrhage and exudates. May be visual loss.

5- Malignant hypertension causing acute microvascular damage (necrotizing vascular tesion) with retinopathy and papilloedema
Microangiopathic hemolytic anemia and D.I.C. occurs in severe cases.

Treatment:

Hypertensive emergency:

Treatment rules:

- Rapid reduction in B.P. is unnecessary it causes hypoperfusion as auto regulation is lost.
- B.P. lowering of 25% over 1-4hr. with a reduction over 24hr to diastolic B.P. of 100mmHg.

The only 2 situations in which BP must be lowered rapidly are aortic dissection and MI.

- Alert well patients may be treated with oral therapy
- Sublingual nifedipine must be avoided

I.V. Drugs used for treatment of hypertensive emergencies

Only used in severe cases

	Action	Dose	Side effect
Sodium nitroprusside	Direct vasodilator starts to act in seconds	0.25-10μg/kg/min infusion	Cyanide toxicity
Nitroglycerine	Vasodilator act in 2-3 min.	1-10mg/hr infusion	Tolerance
Labetalol	Alpha and beta blocker. Best drug in aortic dissection and phaechromocytoma crisis	20 - 80mg I.V. bolus/10min	Avoided in heart failure
Hydralazine	Vasodilator. Used in eclampsia	5-10mg I.V. slowly	May induced angina

Hypertensive urgency:

Treated with oral drugs.

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Hypertensive urgency:

Treated with oral drugs.

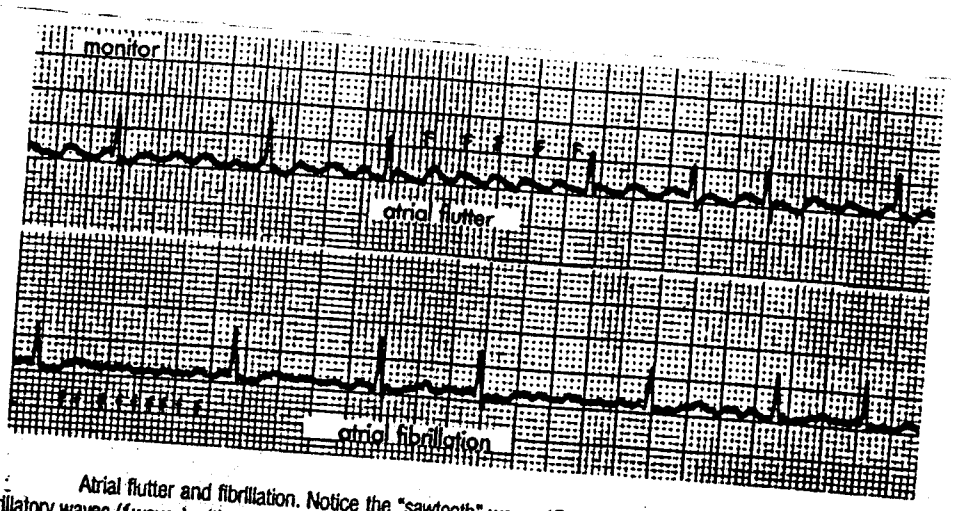
Emergency management of tachyarrythmias

Classification

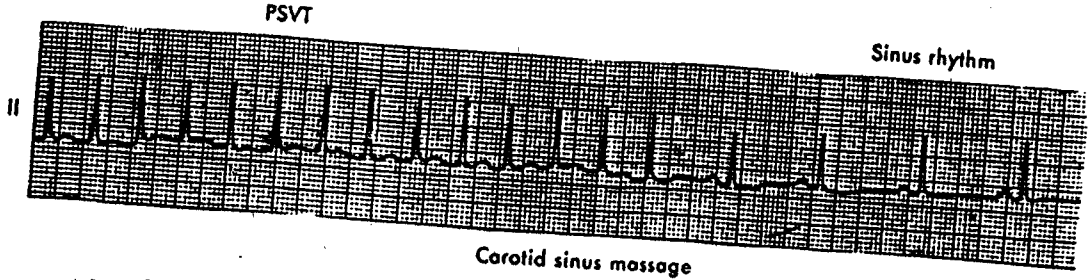
Depends up QRS duration and R.R. intervals

H.R.> 100/min

- QRS<0.12Sec.
  - R.R intervals
    - Regular
      - Sinus tachycardia
      - Atrial flutter
      - A.V. nodal re-ntry tachycardias
    - Irregular
      - Atrial fibrillation (AF)
      - Multi focal atrial tachycardia M.A.F.
- QRS>0.12sec
  - Ventricular tachycardia
  - Supra-Ventricular tachycardia with bundle branch block



Atrial flutter and fibrillation. Notice the "sawtooth" waves (*F* waves) with atrial flutter and the irregular fibrillatory waves (*f* waves) with atrial fibrillation.



Paroxysmal supraventricular tachycardia (PSVT) treated with carotid sinus massage. The first 14 beats in this rhythm strip show the tachyarrhythmia with a rate of about 140 beats/min and no visible P waves. Carotid sinus massage resulted in its abrupt termination, with the appearance of normal sinus rhythm. The PSVT here is probably an atrioventricular nodal reentrant tachycardia (AVNRT).

Atrial fibrillation

Commonest arrythmia

Causes:

- Structural heart disease (ischemic, valvular, cardiomyopathy, pericardial).
- Thyrotoxicosis
- Lone (no heart disease) .
- Pulmonary embolism .

Complications:

- Precipitation of acute heart failure "atrial contraction accounts for 25% of L.V. diastolic blood"
- Formation of left atrial thrombus with systemic embolism.

Acute management:

Lines:

I- ***Cardioversion*** to sinus rhythm:

1- **Electrical:** in hypotension or heart failure. Premedication with midazolam and or/morphia. 200Joules electric shock increased by 100J if no response.

2- **Pharmacological :**
indicated in A.F. less than 2 days without hemodynamic compromise. Recent onset A.F. can convert spontaneously to sinus rhythm.
Ibutilide 1mg I.V. produces rapid response.

II- ***Controlling H.R***. to slow A.F. allowing adequate diastolic L.V. filling by:

a) Calcium channel blockers: diltiazem is the best because it has least –ve inotropic and hypotensive effects.

b) β- blockers:
Esmolol : is ultrashort β- blocker given I.V.
or Metoprolol: 5mg I.V. slowly.
Combined use of I.V. beta and calcium blockers leads to serious negative inotropism

c) Amiodarone : Least negative inotropic effect. Given as 300mg I.V. slowly then I.V. infusion drip in a central line. It slows A.F but can revert it to sinus rhythm.

d) Lanoxin : 0.25mg/d. Esp. In heart failure.

Anticoagulants :
Used to prevent thrombo-embolism especially in high risk patients (e.g. rheumatic valve disease, prosthetic valve, heart failure, ischemic heart).
They should be used 3 weeks before elective cardioversion in A.F. more than 2 days.

Ventricular tachycardia (V.T)

It rarely occurs without structural heart disease. If sustained it causes loss of pulses and consciousness.

Acute management:

D.C cardioversion initially. 100J followed by 200,300, 360J. if necessary
Or

- Amiodarone I.V. 150mg slowly followed by infusion 0.5-1mg/min.
- Xylocaine (lidocaine) 1mg/kg bolus then 2-4mg/min. infusion (may cause seizures in overdose)
- Mg. sulphate 2mg I.V. in torsades De points V.T. characterized by prolonged QT interval above 0.44sec. Caused by electrolyte disturbances or drugs (antipsychotics, antiarrythmics).

Respiratory failure (R.F)

Definition: Clinically it is impossible to predict PO_2 or PCO_2 and diagnosis relies on ABG analysis

Two types:

Type I: Hypoxia (PO_2<60mmHg) with normal or low PCO_2. caused by local lung disease with perfusion of non-ventilated alveoli (shunting of blood without oxygenation). Increasing ventilation of remaining normal alveoli washes out CO_2, however, it cannot correct hypoxia [hemoglobin O_2 saturation cannot increase > 95-100% as O_2 dissociation curve is flap topped]

Lung disease causing type I R.F:

- Pneumonia • ARDS • Pulmonary edema
- Lung collapse • Pneumothorax • Severe asthma
- Pulmonary embolism

Type II: Hypoxia with hypercapnea (PCO_2>49mmHg) caused by hypoventilation in:

- Respiratory center affection: narcotics, sedatives, (low respiratory rate) brain stem stroke. Increased I.C.T.

Neuromuscular:

- Guillian Barre polyneuropathy weakness
- Myasthenia gravis.
- Dermatomyositis, end stage myopathy
- Severe hypokalemia
- Rabies, botulism, tetanus
- Flai chest injury

Respiratory muscles weakness causes low tidal volume and vital capacity.

Chronic obstructive lung disease:

Causes generalized ventilation/ perfusion mismatch

Urgent management of R.F.:

Urgent investigations:

- ABG •CXR
- ECG (tachyarrythmias or ischemia).
- Forced vital capacity and FEV_1
- toxicology screen
- Assessment of respiratory muscles by bedside EMG and muscle enzymes.

Poor prognostic signs (indicating mechanical ventilation):

- Respiratory rate>40 • H.R> 100
- Hypotension or shock • Stridor.
- Signs of respiratory muscles fatigue:
  - Use of accessory respiratory muscles
  - Paradoxical abdominal muscles movement
  - Inability to speak.
- Coma • Pulse oximetry O2 % <90%
- PEF<30% in acute asthma
- Failure of hypoxia to correct to oxygen therapy
- Progressive hypercapnea

Benefits of mechanical ventilation:

Ventilator replaces muscles of respiration allowing their rest [energy source].

They apply intermittent positive inspiratory pressure by pump. Expiration is passive by elastic recoil.

Complications of mechanical ventilation:

- Barotraumas: pneumothora pneumomediasttium, subcutaneous emphysema due to ruptured alveoli
- Hypotension due to decreased venous return from elevated intrathoracic pressure
- Ventilator associated pneumonia
- Stress gastric ulcer.

Acute exacerbation of COPD.

Causes:

- Infection (typically H. influenza, S. pneumonia or viral). [yellow produce sputum]
- Pneumothorax
- Expansion of large bullae
- Excessive sedation by diazepam or opiates.
- Sputum retention with lung collapse
- Pulmonary embolism (common due to secondary polycythemia and D.V.T)

Presentation By deteriorating hypoxia and hypercapnea with disturbed conscious level, features of congestive heart failure, severe bronchospasm

ABG: shows low PH and normal bicarbonate.

Urgent treatment:

(1) **Oxygen therapy:**

O_2 should be prescribed as a during with a writing flow rates or concentrations

Uncontrolled O_2 therapy worsens hypercapnea and level of consciousness controlled O_2 therapy given by a venture mask 24%- 28%. Nasal canulae is unreliable

epeat ABG after 30min. Aim to get O_2 sat ≥92%

Non- invastive nasal intermittent positive pressure ventilation (NIPPV):

Applied by a small machine via a light – fitting nasal mask, delivers positive inspiratory pressure of oxygen. Tit does not need intubation. It is the first – line treatment of choice, as it decrease mortality and allows higher O_2 therapy without rise in PCO_2.

Mechanical ventilation:

Is used in patients who cannot tolerate NIPPV.

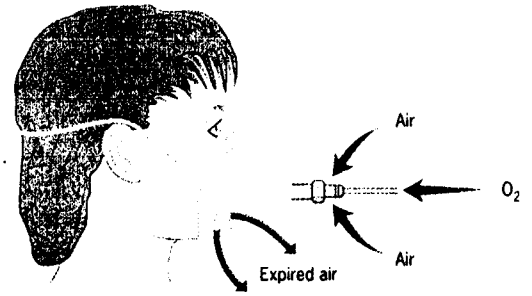
(2) Nebulized bronchodilators [salbutamol 5mg, ipratropium bromide 0.5mg) 14-6hr.

- I.V. aminophylline.

(3)Steroids as anti-inflammatory methyl prednisolon125mg I.V./6hr or 30-40mg/day orally.

(4) Respiratory stimulant by doxapram.

(5)Treat cause of exacerbation: e.g. antibiotics: cefuroxime 150mg/8hr. I.V. with a macrolide.



'Fixed-performance' device for administration of oxygen to spontaneously breathing patients (Venturi mask). Oxygen is delivered through the injector of the Venturi mask at a given flow rate. A fixed amount of air is entrapped and the inspired oxygen can be predicted accurately. Masks are available to deliver 24%, 28% and 35% oxygen

Acute renal failure (ARF) in ICU

Definition :

Abrupt reduction in GFR leading to:

- Rise of serum creatinine > 0.5ml /dl or ≥50% of the base
- Oliguria: < 400ml /day or < 0.5 ml/kg/hr.

Etiology:

I-Pe renal:

1. Hypoxolemia
2. L-ow cardiac output states e.g. end stage heart failure, hepatic failure.
3. Drugs impairing glomerular flow e.g. ACE inhibitors
4. Severe prolonged pre-renal conditions lead to acute tubular necrosis (A.T.N).

II- Renal:

1- A.T.N : (5 0% of all causes) may be :

a. Ischemic: prolonged pre- renal cause.

b. Nephrotoxic

Exogenous toxin: e.g. Iodinated contrast dyes.

Endogenous toxin:

- Hyoglobinuria "rhabdomylosis"
- Hemoglobinuria "intravascular hemolysis"
- Uric acid crystals "tumor lysis syndrome"

c. Sepsis syndrome

In A.T.N there is injury and sloughing of epithelial tubular cells obstructing lumen of tubules

2- Acute glomerulonephritis (GN):

a. Acute post- infection G.N.
b. Lupus crisis
c. Anti- glomerular basement membrane antibody "Good pasture"

3. Acute interstitial nephritis:

Caused by drugs (antibiotics) or infections (severe pyelonephritis).

4. Vascular diseases:

- Malignant hypertension
- Acute vasculitis
- T.T.P.
- Primary antiphospholipid syndrome

III- **post- renal:** obstructive uropathy by enlarged prostate or stone.

Diagnostic approach

1- Exclude obstruction: "corrected urosurgically"

a) • Distented tender bladder: diagnostic bladder catheter and U/S post- Void.
• U/S on Kidneys
• Non- contrast spiral CT abdomen is the best non-invasive method for stone detection

Diagnosis of hypovolemia "corrected by I.V. fluids "

C.V.P.:

- Very low 1-2mmHg
- Hemoglobin saturation in C.V blood <50% indicates low cardiac output states with increased O_2 extraction.

2. **Examination of urine:**
    - Spot urine sodium: in prerenal cases < 20mEq/L " healthy tubules reabsorbs Na" , while in A.T.N. it is >40mEq/L "no reabsorption"
    - Urine osmolarity :> 500 in prerenal and <350 in A.T.N.

Urine sediment:

- In A.T.N: epithelial cell casts, brown casts of necrosed epithelial cells
- In G.N.: active sediment of red and white cell casts
- Positive heme test in urine with no red cells indicates hemo-or myoglobinuria.

4- Exclude end stage chronic renal failure (CRF) presented with oliguria by: Small kidneys by sonar, and renal osteodystrophy in bone X-Ray film in CRF.

New laboratory marker:

Serum cystatin C: found in all nucleated cells. It increases before a rise in creatinine in ARF and not affected by muscle mass.

Emergency management of A.R.F.

1- **Stop all nephrotoxic drugs.**

2- **Correction of life threatening conditions:**

- **Hyperkalemia:** ECG changes are better guide to therapy than serum K. level. It is the commonest cause of death.
- **Hypovolemia:** fluid challenge by 500ml saline or colloid infused over 30min. guided by C.V.P. followed by full replacement.
- **Acidosis:** when serum bicarbonate < 15mEq/L. corrected by I.V. bicarbonate.
- **Uremic symptoms:** (Caused by urea and other solutes):
- Bleeding with hematemesis
- Confusion, asterixis, coma, seizures.
- Pericarditis
- Vomiting
- Anemia
- Arrythmias (from electrolyte disturbances and acidosis).
- Water retention with pulmonary edema
- Hypocalcemia, hyperphosphatemia, hyperuricemia

3. The use of low dose dopamine as a renal vasodilator in acute oliguria is of no benefit. Similarly I.V. frusemide does not increase urine flow as it can not reach it's site of action (intra luminal).
4. infection is the most important cause of death in ARF.

Renal replacement therapy.

Early dialysis when blood urea nitrogen reaches 80-100mg% can avoid occurance of uremic symptoms and allow normal diet. Urgent dialysis is indicated in:

- Volume overload
- Hyperkaemia
- Acidosis
- Uremic manifestations as encephalopathy and pericarditis.

Vascular access:-

Using double lumen large vascular catheter placed in jugular vein or femoral vein allows blood to be pumped to dialysis machine and returned via other lumen.

Intermittent hemodialysis:

Causes rapid clearance of solutes and wastes by diffusion between blood and physiological fluid. It needs high blood flow rate (at least 300ml/min) causing hypotension, and canot be used in hypotensive critically- ill cases.

Hemofiltration:

It removes solutes and wastes by convection (pressure gradient across the semipermeable membrance). It does not need high flow rates, therefore there is no risk of hypotension.

The process is performed continuously. Fluid removed is more physiological than hemodialysis and more suitable in I.C.U. patients.

Electrolyte disturbances

Hyponatremia

Serum Na < 135mEq/L is common in ICU.

Classification

Hypovolemia hyponatremia	Iso volemic hyponatremia	Hypervolemia hyponatremia
Loss of body fluids Renal: • Diuretics • Addisonian crisis • Diuretic stage of acute tubular necrosis • Cerebral salt wasting syndrome Extra renal: Vomiting, diarrhea. pre-renal uremia with rise in serum urea, uric acid	Small gain in free water but not enough to get edema as in: Inappropriate (nonosmotic) release of ADH occurring in brain lesions, pulmonary diseases, neoplasia, mechanical ventilation. Concentrated urine (osmolality >100 mOSm/kg water and low plasma osmolality.	Excess sodium and water gain as in: Hepatic failure Heart failure Nephritic syndrome

Danger of hyponatremia :

Hyponatremic encephalophathy occurs when hyponatermia occurs acutely leading to water shift to brain and cerebral edema causing : headache, vomiting, acute mental changes, seizures, altered consciousness.

Symptomatic cases need urgent correction by saline 0.9% or 3%. Sodium correction should be slow, as rapid correction leads to fatel brain lesion (centeral pontine myolinolysis).

Rate of rise of serum Na should not exceed 0.5 mEq/L/hr. up to 130mEq./L.

Hypernatremia

Serum Na > 145mEq/L

Causes

Hypovolemia hypernatremia	Central diabetes insipidus	Non-ketotic hyperosmolar hyperglycemia
Due to loss of excess water than Na. patients who cannot sense thirst (coma) and do not drink water develop hypernatremia	Occurring in meningitis, head trauma, brain anoxia. Leading to polyuria of dilute urine (Osm <200) corrected by S.C aqueous vasopressin.	Occurs in diabetics under stress (infection) plasma glucose is very high >1000mg/dL causing osmotic polyuria and hypovolemia. ketosis does not occur as there is enough endogenous insulin to prevent it. Treatment: first step is to correct hypovolemia by saline or colloid followed by slow replacement of free water by ½ normal isotonic saline. Insulin is given only after correction of hypovolemia as it drives both water and glucose into cells.

How to calculate plasma Osmolality

Plasma osmolality = 2x plasma Na + $\frac{\text{Glucose}}{18}$ =285mOsm/Kg H_2O

Danger of hypernatremia

It causes brain shrinkage (dehydration) with encephalopathy: Coma, Seizures, focal neurological defect. It should be corrected slowly: serum Na decreases by no more than 0.5mEq/L./hr.

Hypokalemia

Normal serum K: 3.5-5.5mEq/L mainly intra cellular so serum K is insensitive marker of total K stores. Hypokalemia means serum K<3.5

Causes

Transcellular shift of K into cells	Potassium loss	
• Glucose insulin infusion • β- agonists (e.g salbutamol) bronchodilators • Alkalosis • Hypothermia	Renal • Diuretics • Mg depletion impairs K tubular reabsorpation.	Extra renal Diarrhoea

Dangers of hypokalemia

- Diffuse muscle weakness
- It promotes ventricular arrythmias caused by digitalis, ischemic heart.

Treatment :

Mg depletion should be corrected first (it causes referactory hypokalemia). Potassium chloride amp. (2mEq/cm) 20mEq + 100ml saline I.V. (in large central vein to avoid phlebitis) over 1hr. Serum K. rises slowly and takes several days for replacing body stores.

Hyperkalemia

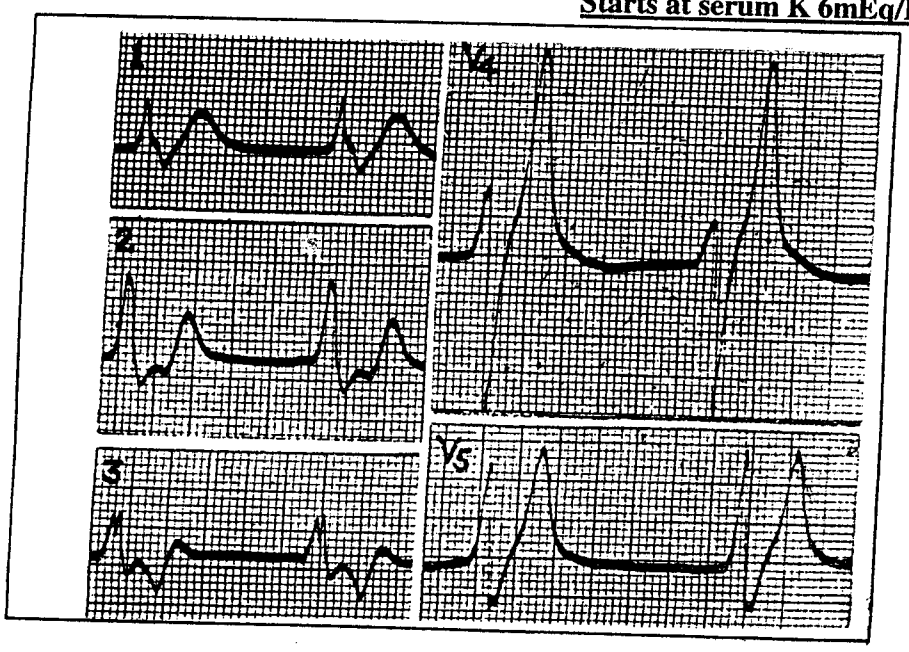
Serum K> 5.5mEq/L

Pseudohyperkalemia occurs form traumatic hemolysis of blood sample

Causes

Renal Failure	Rhabdomyolysis with release of muscle K.	Massive transfusion of stored blood	Drugs blocking aldosterone action
• GFR< 10ml/min. • Oliguria • Diabetic nephropathy damaging renin producing cells with hypoaldosteronism			• ACE inhibitors • Spironolactone • NSAID'S

ECG changes of Hyperkalemia
Starts at serum K 6mEq/L



- Tall peaked T waves
- Absent P waves
- Wide QRS
- Irregular rhythm

Treatment:

1- Calcium gluconate 10% 10ml I.V. slowly antagonizes action of K on heart. Effect only few minutes.

N.B: Calcium is contraindicated in digitalis toxicity causing hyperkalemia by transcellular shift as it potentiates cardiotoxicity

2- 10 units regular insulin in 500mL 20% glucose I.V. over 1hr drives K intracellular.

3- Sodium bicarbonate has the same effect in acidosis

4- I.V Furosemide

5- Hemodialysis.

Hypomagnesemia

Serum Mg is 1.4-2mEq/l (represents only 1% of total body Mg).

Mg depletion the commonest under diagnosed electrolyte abnormality in ICU.

It occurs in diuretic therapy, diarrhea, diabetic ketosis, malnutrition, and myocardial infarction.

It is often accompanied by

HYPO

- Kalemia inhibits K reabsorption
- Calcemia (impairs parathormone release and action)
- Phosphatemia: phosphate deficiency enhances renal Mg loss

Dangers

- Heart: Arrythmias including ventricular tachycardia: torsades de pointes
- Nervous system: Seizures, Coma

Treatment by: Mg sulphate 50% (5gm + 500mL Saline) over 3hr

Metabolic acidosis

Normal ranges:

- PH 7.36-7.44
- PCO_2 36-44mmHg
- Bicarbonate 22-26mEq/L
- In metabolic acidosis

PH and bicarbonate are low. PCO2 is also low (compensatory hyperventilation)

Anion gap:

The measured cation routinely is Na and anions are chloride (CL) and bicarbonate (HCO_3).

Anion gap=unmeasured anions - unmeasured cations
(e.g Sulphate, phosphate, acids) (e.g. Ca , K, Mg)

Which equals: $Na - (CL+HCO_3)$

Normal range : 12± 4mEq/L

High anion gap metabolic acidosis: Occurs by addition of acid to blood resulting in low HCO_3

Causes:

1- Lactic acidosis
2- Ketoacidosis
3- End stage renal failure
4- Toxicity by methanol, ethylene glycol, salicylates.

Lactic acidosis

Lactate is produced by anaerobic glycolysis in:

1- circulatory shock (tissue hypoxias).
2- sepsis (mitochondria toxicity).
3- Thiamine deficiency.
4- Drugs (metformin, propofol, nitroprusside)
5- Enhanced lactate production by muscles in acute asthma and seizures

Diagnosis; Blood lactate >2mEq/L

Ketoacidosis:

Causes :

1- diabetes
2- Alcohol toxicity
3- Starvation

Diabetes Ketoacidosis

Usually precipitated by infection or stress (M.I)

Investigations:

- Hyperglycemia: may be mild
- Urine and plasma ketone bodies are only sensitive for detection " of acetoacetate and acetonebut not β-hydroxybuterate which is the predominant ketone
- ABG's: plasma bicarbonate <12mEq/L indicates severe acidosis
- Urea and electrolytes
- ECG
- Septic screen

Clinical features of diabetic ketoacidosis	
Symptomas : ▪ vomiting	▪ polyuria ▪ abdominal pain
Signs: •Dehyration •Hypotension ▪Air hunger "kussmaul breathing ▪Smell of acetone ▪Drowsiness, coma	

Management :

1- I.V. fluid: fluid deficit 4-8L. (osmotic diuresis) 0.9% saline I.V. 1 liter over 30min, 1 liter over 1 hr , 1 liter over 2 hr ., 1 liter over next 2-4hr.

When blood glucose < 270mg/dl:

Glucose 5% 1liter/8hr (to correct water deficit and to prevent cerebral edema).

2- Insulin:

50units soluble insulin in 50ml 0.9% saline I.V. by infusion pump 6U./hr. initially then 3units /hr when blood glucose < 270mg%.

Blood glucose is checked every hour. If blood glucose does not fall, dose of insulin is doubled.

3- Potassium: plasma K. is initially high (transcellular shift) but falls by therapy . KCL 20-40mEq. over 1 liter fluid at a rate 20mEq/hr

4- Bicarbonate: is only used when PH<7.
Sodium bicarbonate 300mL 1.26% over 30min. in large vein. Complete correction of acidosis should not be attempted.

Additional procedures:

- Urinary catheter
- Nasogatric tube in vomiting or in unconscious patient
- C.V.P and ECG monitor. Plasma expander if hypotension is not corrected
- Antibiotics in infections

complications of diabetic ketoacidosis
▪ cerebral edema (due to rapid fall of blood glucose) ▪ ARDS. ▪ Thromboembolism ▪ D.I.C ▪ Shock state

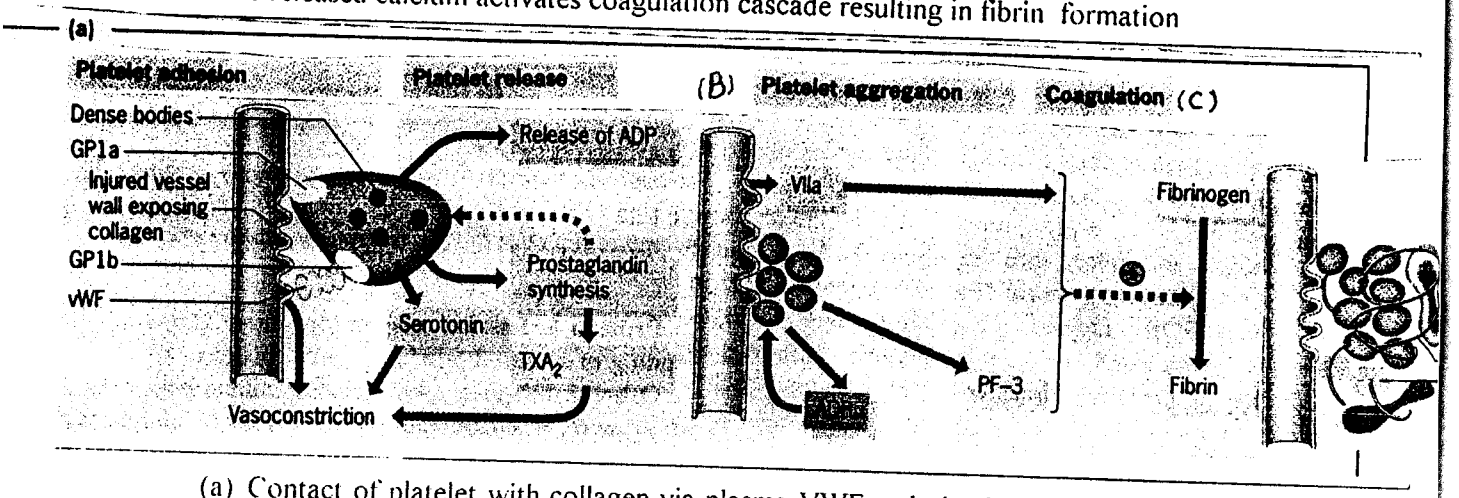
Platelets in critical Illness.

- Normal count 250-400.000/cmm
- Life span 7-10days
- Function: Injury to vascular endothelium leads to platelet adhesion to exposed collagen. Platelets release calcium which activates glycoprotein (IIb- IIIa) receptors on platelet surface. Such receptors bind to:-

a- Von- Willebrand factor (V.W.F.) in the surrounding endothelium which anchors pltaelet plug.

b- Fibrinogen, which bridges platelet resulting in platelet aggregation

The released calcium activates coagulation cascade resulting in fibrin formation



(a) Contact of platelet with collagen via plasma VWF and platelet receptor GPIb activates platelets thromboxome (TXA_2) synthesis and ADP release.

(b) Both TXA2and ADP induces platelet aggregation

(c) Fibrin strands cross-linked by factor XIII binding platelets and red cells.

Thrombocytopenia in the ICU:

(plalelet count <100.000/cmm)

Causes:-

1- Heparin – induced:

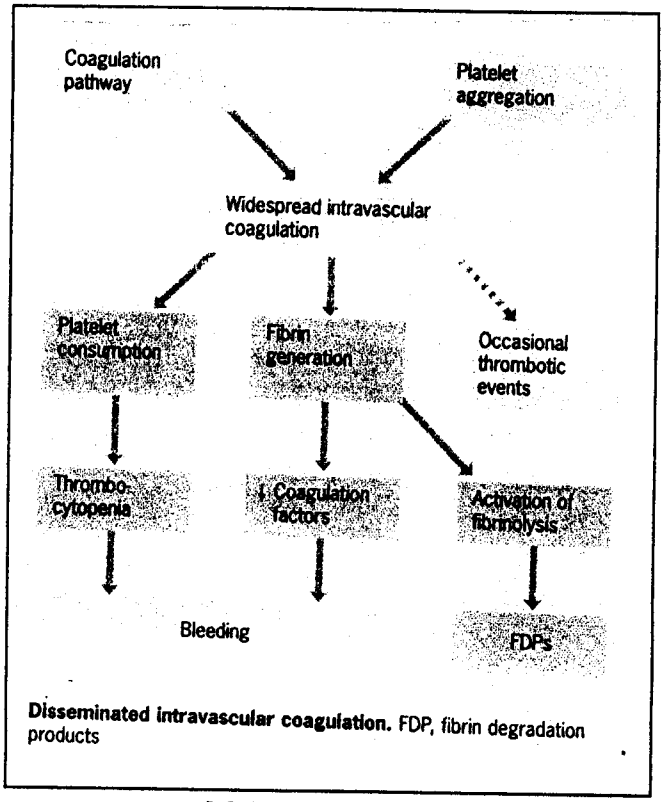
Heparin binds to plalelet factor 4 forming antigen inducing antibodies, which bind platelets together resulting in consumptive thrombocytopenia and clinical thrombosis "due to platelet activation with calcium release and thrombin formation on surface of platelets" Thrombosis may be venous or arterial and occurs 5-10 days after heparin therapy It can occur with very low doses of heparin.

All heparins must be stopped and the patient is treated by direct antithrombin inhibitors. Warfarin should not be used as anticoagulant, as it leads to protein C- deficiency and catastrophic limb gangrene, warfarin induces more rapid fall in protein –C before Vitamin K dependent clotting factors leading to procoagulant state.

2- Disseminated intravascular coagulation(DIC).

Widespread endothelial damage results in release of tissue factor which activates coagulation cascade resulting in:

Microvascular thrombosis and damage of tissues, depletion of platelets and coagulation factors causing bleeding (consumptive coagulation)



Disseminated intravascular coagulation. FDP, fibrin degradation products

Causes: •Sepsis • Multiple trauma

- Obstetric emergencies (abruptio placenta , eclampsia, retained dead fetus
- Malignancy

Clinical features:

- Multiorgan failure (MOF)
- Mucosal bleeding, oozing from intravascular punctures
- Purpura fulminans " in meningococcemia " or any sepsis symmetrical hemorrhagic purpura and necrosis of the limbs

Laboratory diagnosis:

- Low plalelet count.
- Reduced serum fibrinogen <1gm/dl
- Prolongation of P.T. and P.T.T
- Elevated F.D.P's and D.Dimer level
  specific as it represents cleavage
  of fibrin – fibrin bounds)
- Treatment:

1- Treatment of underlying disease (sepsis)
2- Cryoprecipitate 10μ provide 2.5gm fibrinogen. and fresh frozen plasma
3- Platelet transfusion
4- Heparin is usually ineffective in preventing microvascular thrombosis due to depletion of anti thrombin –III.

3- **Thrombotic thrombocytopenic purpura: TTP:**

It is an immune mediated platelet aggregation with widespread microvascular thrombosis. " as in DIC, classified as thrombotic microangiopaties". It is caused by antibodies against normal VWF- cleaving protease, causing high blood VWF leading to increasing platelet adhesion and clearance without activating coagulation cascade (both PT and PTT are normal)

Clinically:- pentade (fever, acute renal failure, nervous manifestations as coma, seizures, thrombocytopenia and microangiopathic hemolytic anemia with schistocytes in blood film).

Treatment :

Plasmapharesis, which removes plasma containing antibodies to be replaced with fresh frozen plasma, having normal cleaving protease. This is repeated daily for 1 week

4.Massive blood transfusion of 1.5times the blood volume results in dilutional thrombocytopenia as plalelets are lost 24hr after blood storage.

5.HELP syndrome:

[Hemolysis, elevated liver enzymes, low platelets is a thrombotic microangiopathy associated with pre eclampsia (accompanied by hypertension).

6.**Catastrophic antiphosholipid antibodies syndrome :**

It is characterized by any or all of the following 3 manifestations.

(1) Recurrent arterial and or/ venous thrombosis . "multiple thromboses including stroke, coronary, mesenteric, lower limb vessels "

(2) Thrombocytopenia (due to platelet clearance).

(3) Recurrent spontaneous abortions.

Plus positive tests for antiphospolipids antibodies including:-

1- lupus anticoagulant: prolonged P.P.T.
2- anticardiolipin antibodies
3- plasma anti B_2 Glycoprotein I antibodies: carries a high risk of thrombosis. Other possible features are: cardiac valve abnormalities, skin ulcers, livedo reticularis , and hemolytic anemia. Microthrombotic renal disease may lead to renal failure. APA may be primary or associated with lupus erythematosis.

Treatment:

1- heparin and warfarin with INR=3 to 4.5
2- I.V. steroids and immunosuppression with cyclophosphamide
3- Plasmapharesis.

Abnormal platelet function :

(with normal count)

Causes :-

1- Renal failure with impaired platelet adhesion (abnormality in VWF). Bleeding time (B.T.) is prolonged. Hematemesis is the leading cause of death in A.R.F. Dialysis and desmopressin (vasopressin analogue) corrects B.T.
2- Drug induced: Antiplatelets, colloides plasma expanders, non- steroidal anti inflammatory, B. lactam antibiotics

Platelet transfusion :

Platelets from several donors suspended in 50ml plasma can be stored for 7 days, given in the form of 4-6units (200-300ml).

Each unit raise plalelet count by 5000/cmm, an effect apparent after 1hr, lasts for 8 days.

Platelet refractoriness occur after multiple transfusions , and this problem can be avoided by :

- Transfusing ABO compatible platelets.
- Removing leucocytes from plasma.
- Using single- donor platelet transfusion

Indications;

- Active bleeding with platelet count < 100.000/cm.
- Prophylactic if plalelet count is < 10.000/ cmm

Contraindication:

Both T.T.P and heparin- induced thrombocytopenia as platelets can aggravate thrombosis.

Hepatic emergencies

Complications of liver cirrhosis needing I.C.U. admission.

1- **Spontaneous bacterial peritonitis :**

infection of ascitic fluid in cirrhosis causes fever, abdominal pain and tenderness or may be silent and presents with encephalopathy and/ or renal failure.

Diagnosis: Ascitic fluid polymorphs >250/L and culture.

Treatment;

- I.V. albumin improves short term survival and decreases renal dysfunction.
- I.V. third generation cephalosporin "cefotaxime"
- Prophylactic norfloxacin 400mg/day to prevent recurrence.

2- **Hepato renal syndrome :**

It complicates advanced cirrhosis and ascites in 10%. kidneys are structurally normal, but there is severe renal cortical vasoconstriction.

Diagnosis:

- Raised urea, creatinine
- Oliguria
- Normal urine sediment
- Normal sonar kidneys
- Exclusion of hypovolemia which causes pre- renal failure and often due to over-diuresis or large volume paracentesis

Treatment:

- I.V. albumin infusion with terlipressin (Glypressin).
- T.I.P.S.
- Liver transplantation.

3. **Pulmonary complications:**

a) Hepatopulmonary syndrome:

Intra pulmonary vascular dilatation causes right to left shunt, resistant hypoxaemia "especially on standing", cyanosis, clubbing. Shunting can be detected by contrast ECHO. Treatment of choice is liver transplant

b) Portopulmonary hypertension:

Pulmonary hypertension due to vasoconstriction of pulmonary arteries causing dyspnea

4. **Hepatic encephalopathy :**

Etiology: liver failure and portosystemic shunting of blood carrying "biochemical neurotoxins" produced in the gut and not metabolised by the liver

Clinically: Early manifestation: (grade I) is very mild, difficult to detect including:

- Sleep rhythm disturbance.
- Poor mentation
- Personality changes
- Slurred speech
- Flapping tremors (asterixis)

Later stages: (Grade 4):

- Coma
- Myoclonic convulsion.
- Hyper reflexia with extensor planter reflex.
- Rarely focal neurological signs
- Fetor hepaticus "bad odour of smell"

Investigations: Diagnosis in mainly clinical

- Arterial ammonia is raised but of no diagnostic value
- EEG shows non-specific changes.

Treatment:-

1- Removal of precipitating factors:
- GIT bleeding
- Constipation
- Infection
- CNS- depressant drugs (diazepam , morphia).
- Diuretics induced hypokalemia, hypovolemia , uremia.

2- Suppress production of nitrogenous neurotoxins by bacteria in the bowel
   a. Dietary protein restriction is no longer recommended as it worsens nutrition in already malnourished cirrhotics
   b. Lactulose 15-30mL /8hr. orally to cause bowel motion twice daily. It produces osmotic laxative effect and reduces colonic PH trapping ammonia in colon preventing its absorption
   c. Oral neomycin 1-4 gm/4-6h., metronidazole and rifaxamin are antibiotics reducing colonic becteria
      Neomycin is poorly absorbed, however it is contraindicated in uremia

3- Cleansing enemas

5- Variceal hemorrhage:

Occurs when portal pressure > 10mmHg
Mortality 50%

Source:
- Large esophageal varices.
- Congestive gastropathy.

Effects
- Shock
- Impaired hepatic functions
- Hepatic encephalopathy
- Sepsis " ciprofloxacin is given for all cases"

Treatment :

1- Hemodynamic resuscitation with colloids/ blood / fresh frozen plasma .
   Air way protection. Ventilator if needed

2- Urgent upper endoscopy:

Value:
- To detect source of bleeding. About 20% of patients have bleeding acute gastric erosion and not varices.
- Banding or sclerotherapy:
  Stops bleeding in 80% of cases and can be repeated.

3- Sengstaken balloon tamponade:
   Only used as a temporary measure when endoscopy fails

4- Drug reduction of portal pressure:
   Is less effective, and is not always used
   a. Terlipressin 2mg I.V. /6hr "vasopressin analogue"
   b. Somatostatin "Octreotide " 50μg I.V. followed by infusion 50μg /hr is a mesenteric vasoconstrictor.

5- Transjugular intra hepatic porto systemic stent shunt TIPSS).
   It is more effective than endoscopic treatment, but it does not improve survival and is associated with more enpcephalopathy. It is used to stop bleeding not responding to endoscopic therapy

6- Surgery: Oesophageal transection combined with splenectomy has high mortality and only used when all therapy fail.

Fulminant hepatic failure (FHF).

Definition :-

Onset of encephalopathy within 8 weeks of jaundice in patients with acute hepatic injury and no previous history of cirrhosis.

Etiology:

1- viral hepatitis A,B,D.
2- drugs (e.g paracetamol), halothane or mushroom toxins.
3- Other causes: leptospirosis, shock and heart failure, Budd- chiari syndrome, acute fatty liver of pregnancy.
4- Cryptogenic "no cause or non A-E viruses"

Diagnosis:

a. Hepatic failure with Jaundice and encephalopathy. The liver is normal or small is size (massive necrosis)
b. Coagulopathy.
c. Cerebral edema with unequal or fixed pupils, hypertensive episodes, bradycardia (reflecting increased I.C.T), myoclonic seizures and rarely papilloedema.

Treatment :

Meticulous supportive treatment in ICU improve survival.

Encephalopathy :-

It differs from encephalopathy of cirrhosis in:

- Only respond to therapy when liver functions improve
- Often associated with hypoglycemia and cerebral edema

Lactulose is given via nasogastric tube or rectally

Intubation to protect air way and allow ventilation

Cerebral edema:-

Diagnosed by measuring I.C.T.C.T brain is not diagnostic.

Goal to maintain I.C.P< 20mmHg by head elevation , control agitation, I.V. mannitol, and hyperventilation (PCO_2 25-30mmHg).

Hypoglycemia: Due to impaired gluconeogenesis treated by10% Glucose I.V. injection

Coagulopathy: Vitamin K., fresh frozen plasma.

Stress ulcer bleeding: I.V. omeprazole

Agitation: Caused by hypoxia, encephalopathy cerebral edema, sepsis [sedation]

Infection: is the leading cause of death: frequent cultures, empirical antibiotic.

Hepatic transplantation should be urgent before complication occur., it improves survival. Without liver transplant mortality is 80%

Endocrinal emergencies

Acute adrenal failure (addisonian crisis)

Etiology	Clinical picture	Treatment
1- Hemorrhage with anticoagulants 2- Infarction in sepsis especially meningococcal septicaemia 3- Sudden withdrawal of steroids or stress (surgery, M.I) in patients on steroids.	1- Refractory hypotension to vasopressors. 2- Hypovolemia and dehydration 3- Hypoglycemia hyponatremia, hyperkalemia 4- Vomiting, diarrhea.	1- Saline infusion guided by C.V.P. 2- Glucose infusion in hypoglycemia" 3- Hydrocortisone 50-100mg I.V./6H. then orally.

Relative adrenal insufficiency occurs in sepsis syndrome and only diagnosed by the rapid ACTH stimulation test (post ACTH plasma cortisol does not rise).

Empiric trial of steroids should be used in clinically suspected cases before plasma cortisol assay

Myxedema coma

Clinical picture	Diagnosis	Treatment
• Hypothermia • Pericardial effusion • Myopathy with raised muscle enzymes • Hyponatremia, hypoglycemia • Hypoventilation with type II respiratory failure.	. elevated blood T.S.H.	• oxygen (may be ventilator) • gradual rewarming • I.V. or oral T3 25µg/12h (acts more rapidly than T4) • Hydrocortisone 100mg I.V./8hr (for associated adrenal failure) • Glucose infusion for hypoglycemia.

Thyrotoxic crisis

Etiology	Clinical picture	Treatment
• Thyroid surgery in unprepared patient • Stress in thyrotoxic patients • Immediately after destructive iodine therapy	• Hyperpyrexia • Coma, agitation • Hyperdynamic shock • A.F. • Hypovolemia from vomiting, diarrhea and excessive sweating.	• I.V. propranolol 1mg/5min. • I.V. fluids • Hydrocortisone 100mg I.V. /8hr. "thyroid crisis accelerates steroid catabolism). • Lugols iodine 4 drops/12hr. blocks thyroxine release. • Oral antithyroid drugs. • Plasmapharesis.

Acute hypercalcemia

Etiology	Clinical picture	Treatment
• Hyperparathyroidism • Malignancy	Occurs when serum Ca> 14mg/dl • Vomiting, peptic ulcer with Hematemesis, ileus • Pancreatitis • Hypovolemia • Polyuria • coma	• Saline infusion to correct hypovolemia • Frusemide I.V. 40-80mg/2hr. to remove calcium in urine • Calcitonin 100U/6hr/I.M./S.C • Hydrocortisone 50-100mg I.V./8hr. • I.V. bisphosponates inhibits bone resorption • Dialysis.

Acute hypocalcemia

Etiology	Clinical picture	Treatment
• Low ionized Ca (normal 4-5mg/dl) • Alkalosis • Renal failure. • Pancreatits • Mg depletion (reduces para thormone release and action). Blood transfusion (citrate in blood binds Ca) • Hypoparathyroidism.	• Tetany • Seizures • Hypotension • Ventricular arrythmias	• I.V. Calcium. Gluconate 10% in 100ml saline infusion over 10min.

Hypoglycemia

Etiology	Clinical picture	Treatment
• Insulin or sulphonylurea over dose • Fulminant hepatitis. • End-stage renal failure (absent gluconeogenesis) • Sepsis. • Adrenal, thyroid, pituitary in sufficiency • Alcoholism • Drugs	Occurs when plasma glucose is < 50mg/dl 1- Adrenergic symptoms as tachycardia, sweating tremors. 2- Neuroglycopenia: seizures, slurred speech, confusion , coma, reversible neurologic focal defects.	• I.V. 50ml of 50% glucose followed by glucose infusion to avoid recurrence • Glucagen or somatostatin analog

Treatment of diabetes in critically ill ICU patients.

Is by continuous I.V. infusion of regular insulin using infusion pump. S.C. injection has unreliable absorption. Sliding scale of boluses of regular insulin should be avoided. Metformin should be stopped (causes lactic acidosis)

Goal : strict control of plasma glucose (not greater than 150mg/dl) improves mortality in patients with sepsis, myocardial infarction and stroke. Never stop insulin infusion in critically ill (as low as 0.5-1U/hr. can be used) as ketoacidosis can occur rapidly.

Rheumatological Emergency

Vasculitis

Definition: inflammation and necrosis of blood vessel wall leading to thrombosis /infarction of tissue or perforation and hemorrhage.

Classification :

Vessel size	Vasculitis
Large	• Takayasu arteritis • Giant cell (Temporal) arteritis (GCA).
Medium	• Polyarteritis nodosa (PAN)
Small	Immune complex mediated: • Hypersensitivity vasculitis • Cryroglobulinemia • Henoch- Schonlein purpura ANCA positive: • Wegners granulomatosis (W.G). • Microscopic polyangis (M.P.A.) Churg-Straus vasculitis (C.S.V.)

Clinical presentations

No single presentation. It has several mimickers of multisystem diseases. Acute presentations include:

1- Acute visual loss, scalp and temporal artery tenderness with diminished pulsations in GCA.
2- Absent radial pulses (pulseless disease), stenosis of aorta and its branches (with acute stroke or M.I) : Aortic arch syndrome and asymmetrical B.P. in takayaus arteritis.
3- Hypertensive crisis in PAN " renal artery stenosis" and acute arterial vascular occlusion of cerebral, coronary, mesenteric arteries
4- Pulmonary- renal syndrome: rapidly progressive glomerulonephritis with renal failure and pulmonary haemorrhage with hemoptysis and respiratory failure. It occurs in W.G., MPA and C.S.V.
   This syndrome also occurs in S.L.E. and Good Pastures syndrome
5- Mononeuritis multiplex: asymmetric multiple peripheral and cranial nerve paralysis.
6- Skin lesions: Palpable purpura, punched out ulcers, digital gangrene and livedo reticularis
7- Constitutional symptoms and fever: patients with vasculitis feel sick

Investigations.

1- Tissue biopsy (e.g- skin, temporal artery). Is the most important diagnostic test
2- **Imaging:**
   - Non- invasive CT and M.R. angiography
   - Duplex ultrasound of extracranial vessels
   - Echocardiography and ECG if suspected coronary affection.
   - Angiography
3- **Laboratory:** Acute phase reactants:
   - ESR> 100mm/hr and C- reactive protein >10mg/dl suggests vasculitis if bacterial infection and widespread cancer is excluded
   - Thrombocytosis
   - Elevated serum fibrinogen, ferritin, complement
   - Low serum albumin (negative acute phase reactant).

Immunological: Measurement of antineutrophil anticytoplasmic antibodies in ANCA associated vasculitis

Treatment:

Steroids and immunosuppressive drugs.

Behcets disease:-

Small vessel vasculitis and veins affection with recurrent orogenital ulcers, uveitis , D.V.T and P.E., and CNS vasculitis causing pontine lesions. No diagnostic laboratory marker. Positive pathergy test (sterile pustule at site of skin needle puncture).

Emergency in rheumatoid arthritis .

I- Local in Joints:

1- septic arthritis
2- ruptured bursa (bakers cyst).
3- Spinal cord compression (upper cervical spine sublaxation).

II- Systemic:-

1- Cutaneous vasculitis with ulcers, gangrene
2- Systemic necrotizing arteritis (mesenteric causing bowel infarction)
3- Eye affection leading to blindness.
4- Obstructive bronchiolitis

III- side effects of drug therapy.

As pancytopenia, hepatitis, infections.

Lupus crisis

Severe form of systemic lupus erythematosis, necessating admission to I.C.U.

Clinical manifestations:

Renal : rapidly progressive glomerulonephritis

Neurologic:

- Lupus cerebritis : Psychosis, seizures, coma
- Vasculitis : stroke.

Pulmonary: Alveolar hemorrhage with hemoptysis and hypoxia.

Cardiac :

- Pericarditis
- Myocarditis with heart failure
- Libman – sacks endocarditis with embolism.

Hematologic:

- Thrombocytopenia
- Pancytopenia

Gastrointestinal:

- Acute abdomen due to pancreatitis, peritoneal serositis, mesenteric vascular occlusion

Vasculitis:

- Almost any location (e.g. acute M.I.)

Associated antiphospholipid antibody syndrome:

With multiple arterial and venous thrombosis, pulmonary embolism.

Fever and Cutaneous manifestation.

Treatment:

- Pulse methyl prednisolone 1 gm I.V. daily for 3-5 days. It may cause severe hypokalemia and arrythmias.
- Plasmapheresis for lupus cerebritis and pneumonitis
- I.V. high dose immunoglobulin for severe thrombocytopenia.

Renal crisis in scleroderma.

It is due to vasculopathy. It causes severe hypertension, renal infarcts, rapidly progressive renal failure.

Hypertension is treated by angiotensin conventing drug inhibitors or direct Angiotensin. II blockers.

Extra muscular manifestations of dermatomyositis

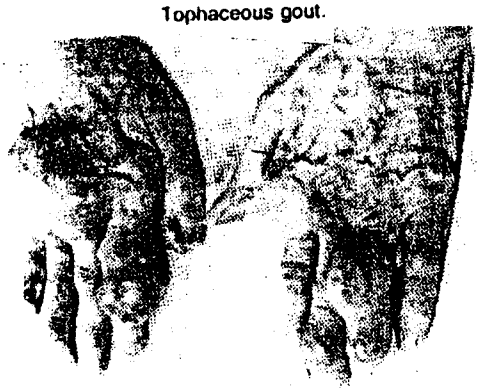
Pulmonary:

- Respiratory failure
- Aspiration pneumonia
- Interstitial lung fibrosis with pulmonary hypertension.

Cardiac: Cardiomyopathy , heart failure, heart block.

Treatment: I.V. high dose steroids

Tophaceous gout.



Acute painful monoarthritis

Causes:

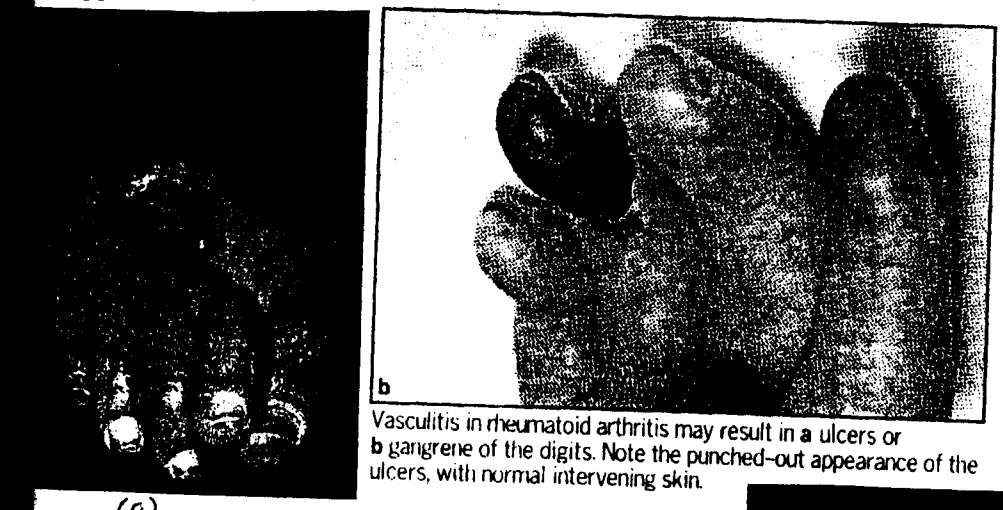
1-Acute hemarthrosis.
2- Acute Gout.
3- Septic arthritis

Joint aspiration: with examination of synovial fluid is diagnosis. Sepsis is diagnosed by W.B.C. > 3000/cmm with gram stain for organism detection. Gout is diagnosed by detection of needle shaped crystals of urate under polarized light microscopy.

Treatment of acute Gout:-

1- NSAID's: Diclofenac 75-100mg I.M. Indomethacin 75mg I.M
2- Colchicine: 0.5mg/huntil sympotoms crase or a maximum of 10 tab. Or GIT toxicity occuns.
3- Corticosteroids I.M./I.V./intra articular
4- Synthetic ACTH. (Synacthen) 1mg I.M./12 for 3 doses

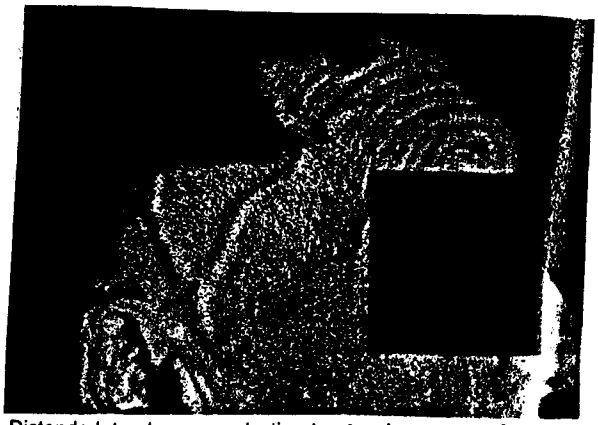
N:B: Allopurinol (Zyloric) should never be started in acute attack. Nor should they be stopped if acute gouty attack occurs while pateint on this medication.



(a)

b

Vasculitis in rheumatoid arthritis may result in **a** ulcers or **b** gangrene of the digits. Note the punched-out appearance of the ulcers, with normal intervening skin.



, Distended, tender, non-pulsating temporal artery due to giant cell arteritis.

Neurological emergency
Coma

Definition:

Consciousness has two components:

Arousal (wake fullness, eye opening) and awareness (responsiveness).

Coma has absent arousal and awareness.

Coma resembles:

- **Persistent vegetative state:**

Wide spraed cortical damage (after cardiac arrest) the patient has intact ascending reticular activating formation (ARA), with normal arousal (eye open) but unawareness, spontaneous non-purposeful movement occurs.

- **Locked in syndrome:** Pontine damage (stroke) with absent all motor activity except up- down eye movements. The patients is awake and aware
- **Brain death:** Cessations of all brain functions

Causes of coma:

I- Diffuse bilateral cerebral damage with intact brain stem occurs in

1- Metabolic encephalopathy states
Organ failure: hepatic, renal, respiratory failure
Enodrinal: hypo-hyperglycemia. Myxedema coma. Addisonian crisis. Pituitary apoplexy.
Hypo- hypercalcemia, hypo-hypernatremia.
Temperature dysregulation : hypothermia. Hyperpyrexia

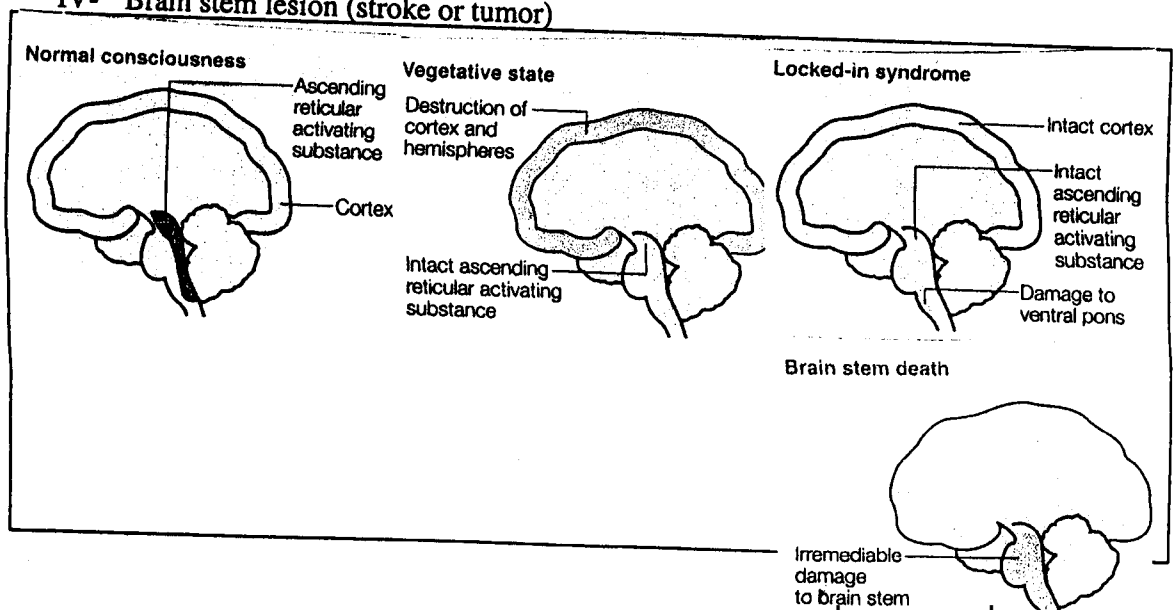
2- Hypoxic encephalopathy
3- Septic encephalopathy
4- Hypertensive encephalopathy
5- Toxic encephalopathy (alcohol, morphia, carbon monoxide)
6- Status epilepticus.
7- Encephalitis, meningitis
8- Subarachnoid hemorrhage
9- Closed head trauma.

II- Supratentorial mass lesion (e.g. massive infarction or hemorrhage) causing:-

a. Midline shift with compression of contralateral cerebral hemisphere.
b. Transtentorial herniation with brain stem compression causing ipsilateral oculmotor nerve palsy (unilateral fixed dilated pupil) and contralateral hemiplegia.

III- Posterior fossa lesion (cerebellar stroke) causing direct brain stem compression and marked rise of intracranial tension.

IV- Brain stem lesion (stroke or tumor)



Bed side coma evaluation

General examination:

Temp: for hypo-hyperthermia.

Respiration pattern

Cheyne-stoke: alterating hyperpnoea and apnoea occurs in metabolic comas. It may be a sign of incipient brain herniation. Kussmaul breathing: occurs in acidosis deep and rapid similar to central pontine lesion breathing:

Ataxic: shallow irregular. Respiratory center damage. Precedes death.

Skin: Jaundice, cyanosis, Rash, purpura, drug injection marks, coarse dry in myxedema.

Neurological examination:

1- Neck rigidity and Brudzinkies reflex (passive neck flexion causes knee flexion) occurs in meningitis.

Missing these signs results in fatal delay in therapy.

2- Eyes:

a) Pupils: (size and light reaction)

- Unilateral fixed: temporal lobe uncus herniation (neurological emergency).
- Bilateral dilated fixed pupils: brain death
- Bilateral pin-point pupils: pontine stroke interrupting sympathetic pathway. Opiate toxicity.
- Normal pupils: metabolic comas and nervous system depression drugs.

b) Ocular movements:

Ocular reflexes: test brain stem function

Oculocephalic reflex: passive head turning produces conjugate ocular deviation to other side (Dolls head reflex).

Caloric test: ice – cold water irrigation of external ear produces eye deviation towards irritated side. Both reflexes are lost in lower brain stem lesion

Abnormalities of conjugate gaze :

- Conjugate lateral deviation occurs towards frontal lobe stroke side and away from hemiplegic side
- Irritative epileptic focal frontal lobe lesion deviates eyes to opposite side
- Ocular bobbing: Sudden brisk downward eye diving occurs in pontine lesion

c) Fundus examination: for papilloedema

3- Lateralizing neurological signs:

Are difficult to elicit in coma. When present, they indicate focal structural brain lesion, and rarely occurs in metabolic encephalopathy.

- Facial nerve palsy: dropping of one side, blowing in and out of paralysed cheek
- Asymmetry of tone, tendon reflexes and planter reflex.
- Painful stimuli causes movement of one side only: flexion movement indicates thalamic lesion (decorticate) while extension response indicates upperbrain stem lesion (decerebrate).

Prognosis of coma

Coma is rarely permenant state.

Glascow coma scale helps in:

1- define coma (scale ≤ 8)
2- indentify candidates for intubation (≤ 8) .
3- prognostic marker:

patients who remain comatosed without eye opening or motor response to pain 1hr. after cardiac arrest have bad neurological recovery in 70-80% of cases.

Urgent coma investigation

1- Full biochemical, endocrinal , hematological screen tests
2- Drug screen
3- Blood culture
4- C.S.F. examination in suspected meningitis or subarachnoid hemorrhage after exclusion of increased I.C.T.
5- Brain imaging
6- E.E.G.

Emergency coma management :

- Support air way and circulation
- Glucose I.V. 50% 50ml treat possible hypoglycemia coma
- Naloxone 0.4- 1.2mg I.V. in suspected opiate over dose
- Flumazenil 0.2mg I.V. in suspected benzodiazepine (diazepam) over dose
- Start I.V. cefatriaxone 2gm immediately in suspected meningitis after taking blood culture

Coma Glascow scale ranges 3 to 15	
Eye opening	Points
Spontaneous	4
To speech	3
To pain	2
None	1
Speech	
Oriented	5
Confused	4
Inappropriate	3
Sound only	2
None	1
Motor	
Obeys commands	6
Localizes pain	5
Withdraw to pain	4
Flexion to pain	3
Extension to pain	2
No movement	1

Diagnosis of brain death

1- deep coma with exclusion of other causes especially hypothermia and narcotic drug toxicity
2- patients is on ventilator because of apnoea
3- Absent all brain stem reflexes with dilated fixed pupils, absent corneal, gag, cough, ocular reflexes.

Tests are repeated 6-24hr before brain death is confirmed.

Confirmatory tests:

1- Iso- electric E.E.G
2- Cessation of brain blood flow detected by transcranial Doppler or angiography
3- Absent stimulatory response of a rise of arterial PCO_2 to 50mmHg to respiratory center when patient is momentaly disconnected from ventilator.

Assessment of acute bacterial meningitis

Presentations:

- Headache, fever, neck stiffness, photophobia
- Confusion, psychiatric disturbances
- Seizures.
- Focal neurological signs indicate complications (abscess, arterial or venous occlusion, raised I.C.T.)
- Meningococcal rash
  - Petechial , correlate with DIC and low platelets
  - Purpura fulminans: purpura with necrosis due to both DIC and vascular occlusion

Immediate management:

1- Blood culture
2- give antibiotics before any investigation (do not delay them beyond 30 minutes) ceftriaxone 2gm/12hr (with vancomycin 500mg I.V./6hr if resistant pneumococci are common).
3- CT scan before lumbar puncture (LP) if sings of increased I.C.T. are present (papilloedema, coma, focal signs, seizures) as brain herniation may occur.
4- L.P. with C.S.F. examination for:

- Neutrophils > 1000/cmm
- Elevated protein >1gm/L.
- Antigen testing
- Low glucose
- Culture: postitive in 50-80%
- Gram stain (neisseria gram –ve diplococci, strep pneumonia gram +ve bacillia)

Prophylaxis of contacts with ciprofloxacin 750mg single dose.

Early management of stroke

Urgent treatment of stroke is essential (Time is brain)

Definition of stroke: sudden onset of a neurological deficit due to focal vascular cause

Causes of stroke

I-**Ischemic 85% :**

Due to acute occlusion of cerebral vessel leading to brain infarction. The tissues surrounding infarct is ischemic but reversibly functioning (ischemic penumbra).

Causes:

1- Cerebral thrombosis of atherosclerotic artery [atherosclerosis risk factors are present].
2- Cardiac embolic (20%):
   - A.F.: (with thrombus in left atrium or its appendage) with rheumatic mitral valve disease.
   - Recent M.I. with transmural thrombus or thrombus in L.V. aneurysm.
   - Mechanical valve thrombi.
   - Vegetations of bacterial endocarditis.
   - Dilated cardiomyopathy.
   - Paradoxical embolic from D.V.T. passing via patent foramen ovale.
3- Artery – to artery embolic stroke:
   Thrombus on top of atherosclerotic plaque in large artery (commonest is carotid bifurcation). May embolize to intra cranial arteries occluding them
   Carotid atherosclerosis produces 5% of ischemic stroke.
4- Small vessel stroke small penetrating artery occlusion causng small lacunar infarction. Hypertension is the risk factor.

Uncommon causes of ischemic thrombotic stroke:

1- System lupus erythematosis with primary antiphospholipid syndrome or liebman sacks endocarditis causing embolism.
2- Large vessel vasculitis
3- Hypercoagulable states
4- Venous sinus thrombosis of brain venous sinuses (postpartum, sepsis, meningitis)

II- **Hemorrhagic stroke (10%).**

1- Hypertensive hemorrhage occurs in basal ganglia, cerebellum, pons.
2- Hemorrhage in subcortical areas (lobar):

Occurs in:

- Congential vascular malformation
- Rupture of mycotic aneurysm
- Anticoagulant therapy and hemorrhagic blood diseases.

Initial assessment of stroke.

Laboratory;

- ABG's
- Serum glucose.
- Biochemical screen
- Hematology and coagulation screen.

Imaging:

Non contrast computed tomographic scans of brain :

- Diagnose cerebral hemorrhage reliably
- Scans in the first few hours after infarction usually show no abnormality and should be repeated 24-28hr. later
- It fails to show brain stem stroke and lacunar infarcts.

CT angiography : after administration of I.V. iodinated contrast to visualize extra and intracranial arteries showing site of vascular occlusion.

Magnetic resonance imaging: MRI.

Can detect site of infarction in any brain area, and more early than CT, however less sensitive than CT in detecting acute blood.

Diffusion - weight images is even more sensitive for early brain infarction.

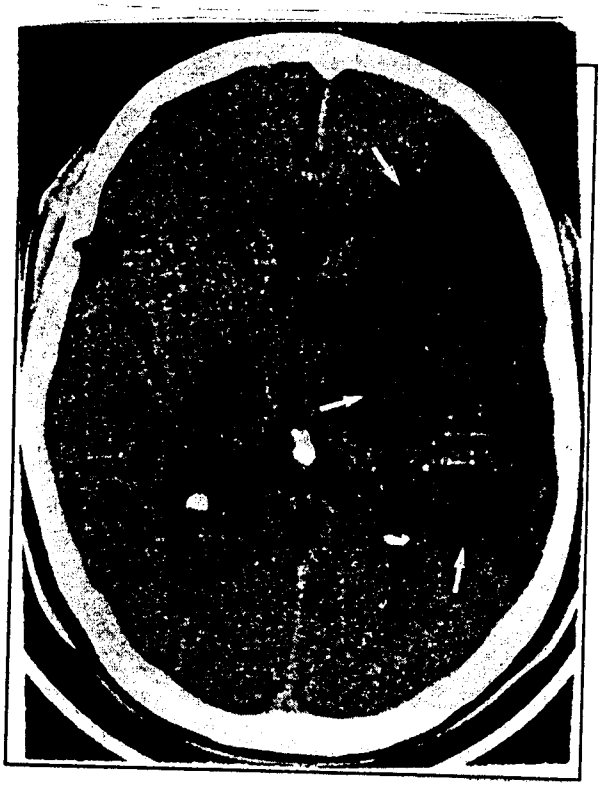
M.R. angiography: is highly sensitive for stenosis of internal carotid arteries and large cerebral arteries

Ultrasonography:

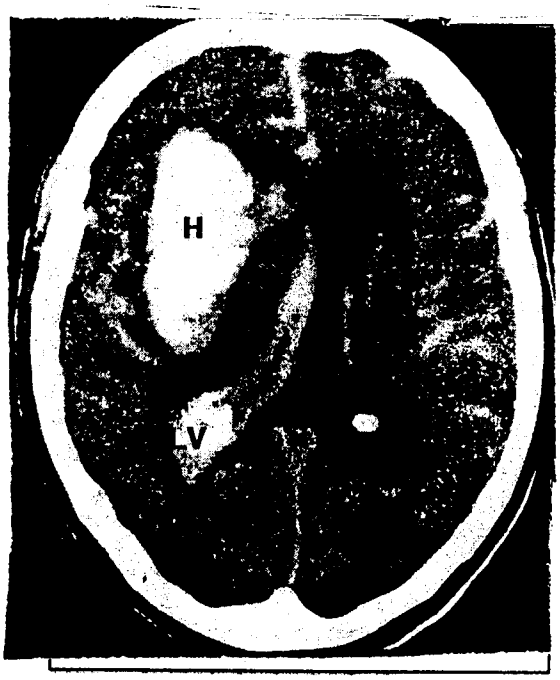
- Duplex scan for diagnosis of carotid artery stenosis
- transcranial Doppler shows high blood flow velocity in stenotic cerebral vessel.

Heart assessment:

- ECG
- ECHo heart for detection of intracardiac thrombus



Unenhanced CT shows low density left cerebral infarction due to middle cerebral artery occlusion



Intracerebral haemorrhage, extended to lateral ventricle and subarachnoid spaces Non-contrast C.T.

H (haemorrhage)
LV(Lateral ventricle)

Treatment of ischemic stroke.

Thrombolysis:

Indications: onset of symptoms ≤3 h. No hemorrhage on CT.

Drug: Tissue plasminogen activator 0.9mg/Kg I.V. (10% as bolus, remainder as I.V. infusion over 1h).

Contraindication:

- Minor stroke or rapidly improving symptoms
- Coma
- Hypertension
- Coagulopathy states.

Results:

Improvement in neurological deficit but not mortality. Incidence of cerebral hemorrhage 6%. Cerebral angiography with intraarterial thrombolytic in clotted artery minimize systemic bleeding.

Antiplatelets: Aspirin 75mg/day alone or combined with dipyridamole or clopidogrel reduces risk of all vascular atherothrombotic events

Anticoagulants :

Thrombotic stroke:

Heparin increases risk of cerebral hemorrhage. It is widely used in stroke in evolution (progressive stoke or transient ischemic attacks) to prevent worsening, with no controlled studies on its benefit.

Embolic stroke:

Warfarin to keep I.N.R range 2-3 used to prevent recurrent embolism. It is used 1-2 weeks) after stroke onset to prevent cerebral hemorrhage.

Long term anticoagulation is needed when embolic source cannot be eliminated.

Medical support:

To optimize cerebral perfusion in ischemic penumbra.

a) Hypoxia: due to aspiration pneumonia (dysphagia iscommon), Coma with upper air way obstruction or associated neurogenic pulmonary edema. Endotracheal intubation, suction of secretions and oxygen therapy.

b) Hypertension: falls spontaneously, and lowering of BP in acute stroke is of no benefit B.P. should only be treated if above 180/120 or in following conditions:

- Malignant hypertension
- associated myocardial ischemia
- Starting thrombolytics .

Both fever and hyperglycemia are deterimental to brain. Blood glucose should be kept <200mg/dLby insulin.

Venous thromboembolism prophylaxis with enoxiparin S.C. or compression leg stocking

Cerebral edema:

Common after large middle cerebral artery infarcts causing increased I.C.T. and transtentorial hernitation.

Treatment by:

- Elevated head bed 30°C.
- Mannitol 250ml of 20% over 15min (water diuresis) (Serum osmolality should not rise>320)
- Frusemide 20-40mg I.V.
- Corticosteroids are of no benefit.
- Hyperventilation by ventilator to wash CO_2 (Kept 25-30mmHg) induces cerebral vasospasm
- Treatment of seizures as it increases edema
- Surgical decompression especially in cerebellar stroke, as it directly compresses brain stem.

- Inappropriate ADH secretions worsens edema and is treated by fluid restriction.

Neuro surgical treatment:

- Carotid endarterectomy for atherosclerotic plaques in symptomatic stenosis of > 70 % or Balloon angioplasty with stenting in high risk surgical patients
- Evacuation of cerebellar hematoma >3cm.
- Acute hydrocephalus due to compression of C.S.F. drainage by blood or edema is treated by ventricular shunting

Status epilepticus

Continuous tonic- clonic convulsion or frequent attacks without regaining consciousness.

Management:

Identify and treat etiology

1- Metabolic encephalopathy:
E.g. ▪ Hypoglycemia ▪ hypocalcemia. Both need urgent correction
- Hypo-hypernatremia (needs slow correction)
- Uremia, sepsis, hepatic failure.

2- Intracranial infection.
3- Stroke.
4- Hypoxia.
5- Cerebral tumor.
6- Anti- epileptic drug withdrawal or non-compliance
7- Drug toxicity (e.g. xylocaine, theophylline, imipenem)or drug withdrawal (alcohol, opiates, diazepam)

Rapid evaluation of new onset seizure

1- Neurological examination showing focal defect neck rigidity:
C.T. brain and lumbar puncture.
2- E.E.G monitoring in patients with prolonged coma to detect nonconvulsive status epilepticus
3- *laboratory studies:*
- Biochemical screen
- Toxicology screen
- Serum antiepileptic drug assay

Identify and treat complications

- Hyperthermia ▪ Rhabdomyolysis
- Hypoxia ▪ Pulmonary aspiration
- Pulmonary oedema ▪ Arrytmias
- Lactic acidosis ▪ Hypertension
- Raised I.C.T. ▪ Hypoglycemia.
- Self injury ▪ Irreversible neurological damage

If seizures stops by correction of underlying factors (e.g. glucose for hypoglycemia) anti-epileptics are not used.

Seizures > 5 min. need urgent therapy to avoid neurological damage or to become refractory.

Management :

Priorities:

Stabilize the patient:

Airway : oral or endotracheal intubation with oxygen

Correct hypotension: with CVP monitor.

Arrythmias: with ECG monitor

Blood sample for analysis.

Anti- epileptics

- I.V. diazepan 10-20mg I.V. (action for 15-30min)
  May cause respiratory depression.
  Lorazepan has a longer duration of action

- I.V. Phenytion (Epanutin)18 mg/kg I.V. infusion on saline at a rate of 50mg/min (e.g. 1 gm over 20min). under ECG monitor (causes arrythmias) and B.P., R.R monitor (causes hypotension, respiratory depression).
- I.V. Phenobarbital 10mg/kg slowly

Refractory status epilepticas:

Needs endotracheal intubation and mechanical ventilation and general anesthesia with:

▪Midazolam ▪ Propofol ▪ Thiopentone under E.E.G monitor

Acute neuromuscular weakness syndromes causing respiratory failure

(1) Myasthenia gravis crisis:

Features: Muscle weakness of eyelid, extraocular muscles, proximal limb muscles, pharyngeal muscles and respiratory muscles.

Several drugs exacerbate myasthenia (e.g garamycin, β-blockers and magnesium)

Treatment:

- Anticholinersterase inhibitor drugs pyridostigmine (Mestinon) 60mg orally /6hr.
- Immunosuppression: prednisolone 1-1.5mg/kgm/day orally (initially may exacerbate weakness). Azathioprine 1-3mg/kg/day.
- Plasmapheresis to remove antibody over 5-7 days.
- I.V. immunoglobulin G (0.4-2gm/kg/day for 2-5days) to neutralize antibody

(2) Guillain- Barre syndrome :

Infectious acute demylinating motor polyneuropathy with progressive symmetric limb weakness and respiratory muscle weakness. Sparing sensations. Associated autonomic instability (tachycardia, arrythmias, swings of B.P). and bulbar weakness. Diagnosed by slow nerve conduction velocity and raised CSF protein.

Treatment: I.V. immunoglobulin G. or plasmapharesis.

(3) Critical- illness polyneuropathy:

Is mixed motor and sensory polyneuropathy complicating sepsis, and diagnosed by nerve conduction studies. No specific therapy.

(4) Muscle diseases causing acute weakness:

Polymyositis: Diagnosed by raised muscule enzymes, E.M.G., and muscle biopsy and treated by steroids.

Hypokalemia :

Acute rhabdomyolysis:

Caused by prolonged convulsions, crush injury, hyperthermia, hypokalemia.

Manifested by dark red brown urine containing myoglobin (but no red cells on microscory), acute renal failure, hperkalemia and raised CPK.

Treatment by alkaline diuresis, rehydration, and dialysis.

Assessment of respiratory muscles:

Respiratory muscle weakness leads to failure to cough, aspiration of secretions with infection and atelecasis and later respiratory failure with CO_2 retension

- Forced vital capacity < 30mL/Kg impairs cough and <15mL/Kg is indication of mechanical ventilation.
- O_2% (by oximetry). PFR and FEV_1 do not correlate with degress of respiratory weakness.
- CXR to detect pneumonia or atelectasis